JUL 7 1927

VOL. LVI. JULY, 1927 No. 4.

ARCHIVES
OF
OPHTHALMOLOGY

FOUNDED IN 1869 BY HERMAN KNAPP

EDITED BY

ARNOLD KNAPP AND WARD A. HOLDEN
NEW YORK

THE DEVELOPMENT OF THE GLAUCOMA SCOTOMA
C. H. THOMASSON

ON THE SUBJECTIVE CYCLOPIC EYE (THE THIRD EYE)
SH. FUNAISHI

A CASE OF TEAR INTO THE CILIARY BODY FROM CONTUSION
H. D. LAMB

A SIMPLIFIED CONCEPTION OF VISUAL FIELD CHANGES IN CHRONIC GLAUCOMA . . . . . LUTHER C. PETER

DIVERGENCE EXCESS: ITS DIAGNOSIS AND TREATMENT
JOHN H. DUNNINGTON

A SIMPLE APPARATUS FOR DETERMINING THE NEAR POINT OF ACCOMMODATION AND THE ASTIGMATIC AXIS . T. P. LEE

FUNDUS CHANGES IN HYPERTENSION WITH AND WITHOUT NEPHRITIS . . . . . AARON BARLOW

(Complete contents inside)

G. P. PUTNAM'S SONS
2 WEST 45TH ST., NEW YORK

PUBLISHED BIMONTHLY SUBSCRIPTION, SIX DOLLARS A YEAR

Entered at the Post-Office, New Rochelle, N.Y., as Second-Class Mail Matter

NOTICE TO CONTRIBUTORS

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with the publication of their contributions.

1. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—**notice of which should be given at the head of the manuscript, or at the latest on the first proof,**—only the additional cost of press-work and paper will be charged to the author. The **number so ordered should be inclusive of the gratuitous copies.**

2. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper. Type-written MS. is preferred.

b. Write without breaks, *i. e.*, do not begin each sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics* should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times; **antique** type should be so marked.

e. Let the title of your paper indicate its contents. If it is a general title, for instance, Clinical Contributions, mention the subject of each special communication,—for instance: CASE I. Sarcoma of Iris; CASE II. Exostosis of Frontal Sinus, etc. These special titles will appear in the table of contents of each number and each volume, under the heading of the general title, so that they will not be overlooked.

f. Illustrations should be carefully drawn on separate sheets.

3. Authors receive proofs for revision, which they should correct and return without delay. We beg, however, to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them to make, if possible, no alterations at all in their MSS., or, at least, to limit them to what is of essential importance.

When sending manuscripts for the ARCHIVES OF OPHTHALMOLOGY, please address the editor, Dr. ARNOLD KNAPP, 10 East 54th St., New York City.

ARCHIVES OF OPHTHALMOLOGY.

THE DEVELOPMENT OF THE GLAUCOMA SCOTOMA.[1]

BY DR. A. H. THOMASSON, NEW YORK.

(With nine figures in the text.)

IN dicussing the diagnosis of non-congestive glaucoma Elliot classes scotometry as the most important procedure of all.[2] He lists the important diagnostic procedures in the following order: scotometry, perimetry, tonometry, ophthalmoscopy, and the testing of the central visual acuity. By scotometry in this connection is meant the careful examination of the blind spot and its vicinity for Bjerrum's sign. He might have added that it is also true that the careful checking up of this sign at frequent intervals gives the most accurate information as to the progress of this disease. By this means may be determined better than by any other whether or not an operation should be resorted to, and if so, when to operate, for though the tonometer may seem to indicate that the intraocular tension is kept within normal limits, there may, nevertheless, be a steady advance in the field of vision that can only be determined by scotometry and checked by operation.

It is now thirty-seven years since Bjerrum first described his sign and pointed out its importance, and yet no detailed

[1] Read before the Section on Ophthalmology, New York Academy of Medicine, February 21, 1927.

[2] Elliot, Col. R. H. "The Diagnosis of Glaucoma." *Transactions International Congress of Ophthalmology*, 1922.

description or illustration of it has appeared in any American textbook on ophthalmology, nor, as far as I have been able to determine, in any American journal of ophthalmology. This surprising lack of interest in so important a symptom is difficult to explain. A partial explanation may be that the early glaucoma scotoma can only be mapped out accurately by Bjerrum's original method, with a very small object at 1*m* or more, and that no convenient apparatus for this work has hitherto been available, also because this scotoma can-

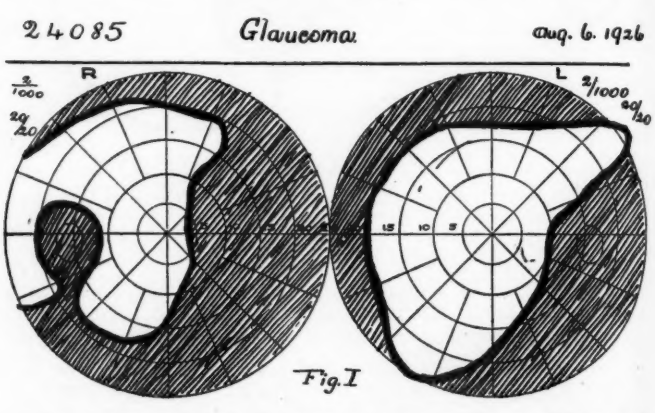


FIG. 1. The field from the right eye shows a single-winged vertical scotoma in which the wing has been extended far beyond the horizontal meridian on the nasal side. The field from the left eye is a somewhat advanced stage of the same type of scotoma.

not be accurately transferred to the ordinary field charts because these are drawn on much too small a scale. This fact was first called to my attention in 1920 by Dr. A. Knapp, who was then using a special field chart of his own design that was drawn on a scale about four times as large as that of the usual chart. It was this suggestion by Dr. Knapp that led to the development of my present field chart and tangent screen. I am also indebted to him because the subject matter of this paper and the fields illustrating it were obtained in the examination of patients of his and under his supervision. All fields here shown are by his permission.

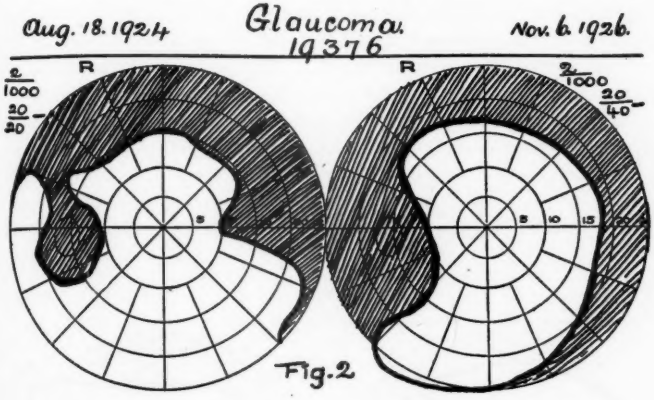


FIG. 2. The left hand field shows a typical single winged vertical scotoma taken August 18, 1924, and at the right is the same field taken two years later, now somewhat advanced.

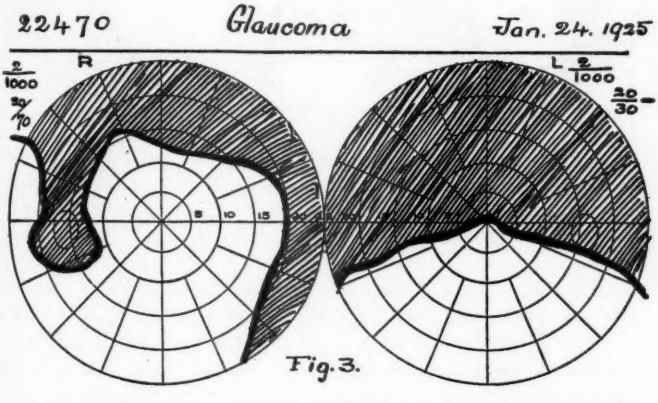


FIG. 3. The field from the right eye shows a moderately advanced single winged vertical scotoma, and from the left eye the same type of scotoma that has advanced to the stage of hemianopsia.

Practically all we know concerning the morphology of the early glaucoma scotoma we owe to the investigations of Bjerrum, Seidel and Roenne. I have paid particular atten-

tion to the descriptions given by each of these investigators and have noted several important differences. Bjerrum's scotoma was sometimes isolated, that is, having no connection with the blind spot. In my experience an isolated scotoma is never an early manifestation of glaucoma, all early scotoma being attached to the blind spot. Late in the disease, however, isolated scotomata do appear in various portions of the remaining visual area. Seidel's scotoma was

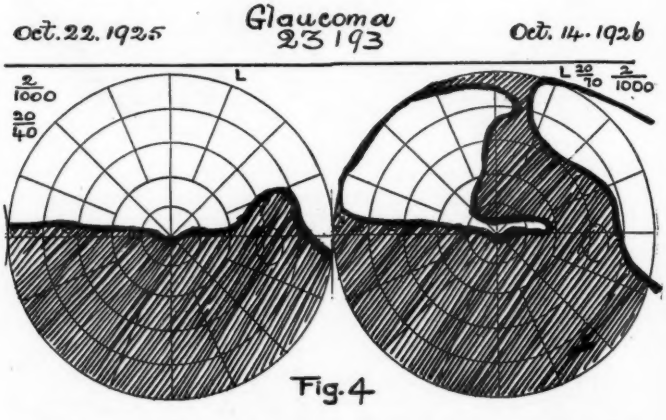


FIG. 4. The left hand field shows the stage of hemianopsia, and the right hand field is from the same eye, but taken one year later. It shows an advancement beyond the stage of hemianopsia. This eye was operated upon in February, 1927, followed by a marked improvement in the field.

always connected with the blind spot, but the extremities of its wings were pointed. This is certainly not in accord with anatomic distribution of the retinal nerve fibers, for they diverge as they pass outward from the optic disc. It was the investigation of this point that led Elliot to the discovery of his sign, which he attributed to the use of the circular method of scotometry. Several of my early scotomata taken with a *2mm* object were pointed, as were Seidel's, but a more careful examination with a smaller object revealed in each case a scotoma that became broader as the distance from the blind spot increased. In some of these only the central stem

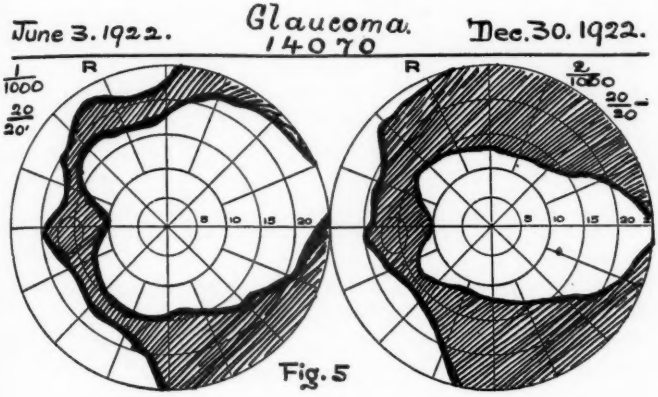


Fig. 5

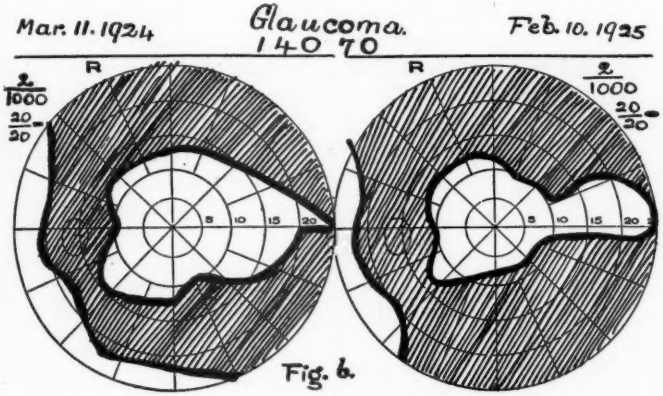


Fig. 6.

FIGS. 5 and 6. These figures illustrate the successive changes in a double winged scotoma. The first was taken June 3, 1922, the last February 10, 1925. The central vision, tension and peripheral field remained practically normal during this time.

could be made out with a *2mm* object, the marginal areas with a *1mm* object, indicating that this central stem, which was pointed, was probably more dense than the marginal areas. This, I think, explains the reason why Seidel's sign was pointed. Roenne showed that when a double winged sco-

toma met on the nasal side each wing ended sharply at the horizontal meridian and that if one wing were more advanced than the other, a "step" was formed. This is what should be expected from the distribution of the retinal nerve fibers, but in many of the scotomata examined by me, especially the one-winged vertical type, there has been an extension far beyond the horizontal meridian on the nasal side. This is contrary to our present conception of the distribution of the retinal nerve fibers, and must be explained on some other hypothesis.

In my series of cases the early glaucoma scotoma appears in three, and possibly four distinct types: (a) single winged vertical, (b) double winged vertical, (c) single winged horizontal and possibly (d) double winged horizontal. In all these type of scotomata the blind spot is at first not enlarged except in the axis of the projecting wings.

The Single Winged Vertical Scotoma.—This scotoma arises from the upper or lower margin of the blind spot, and at its point of origin is about half as wide as the blind spot in its transverse diameter. Its wing is usually broader than the wings of the double winged vertical scotoma. At first its course is almost directly upward or downward, but soon the inner margin curves sharply toward the nasal side, following roughly the 15° circle, and meeting the horizontal meridian on the nasal side somewhere between the 10° and 25° circles.

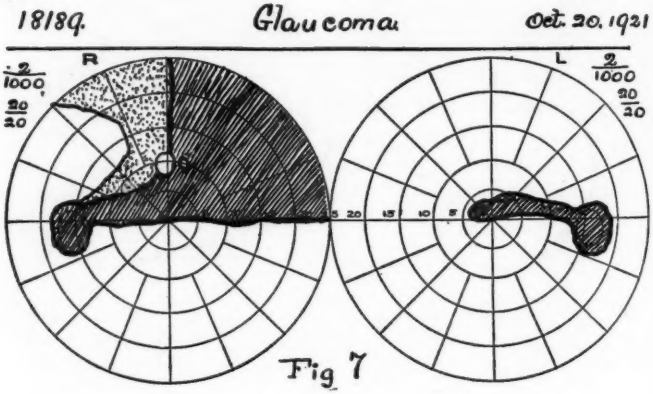


Fig 7

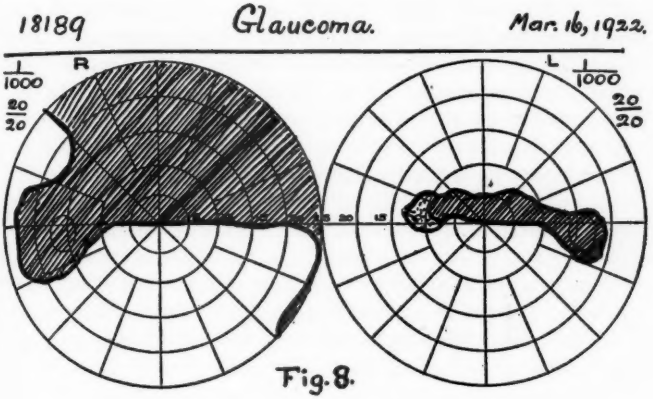


Fig. 8.

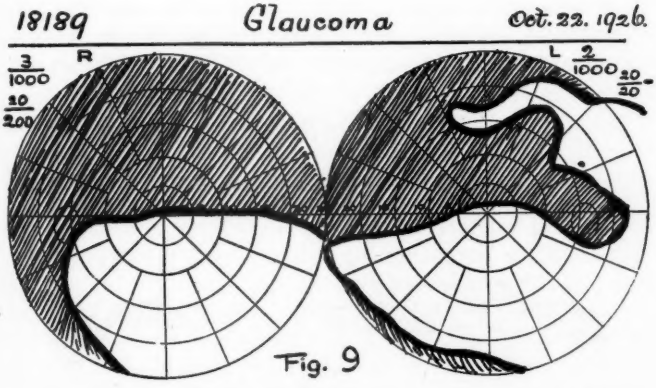


Fig. 9

FIGS. 7, 8 and 9. These fields illustrate the development of a bilateral horizontal scotoma from October, 1921, to October, 1926. The right eye is the more advanced, and in Fig. 9 has reached the stage of complete hemianopsia.

This leaves a semilunar visual area between the scotoma and the horizontal meridian which rapidly fills in from the nasal side. Meanwhile the temporal margin of the scotoma is gradually approaching the horizontal meridian on that side. The result is a vertical hemianopsia, unless a double winged scotoma develops before this stage is reached. After the development of a vertical hemianopsia the other half of the field is usually encroached upon either by the development of another wing from the other pole of the blind spot, which advances as did the first wing, filling in first the nasal then the remaining part of the temporal field; or the first wing may advance beyond the horizontal meridian on the nasal side and from that direction ultimately obliterate practically the entire field.

The Double Winged Vertical Scotoma.—The wings of this scotoma arise from the upper and lower poles of the blind spot. They are at first nearly vertical, narrower than the blind spot (about half its transverse diameter), but as has been stated, as they progress, they are usually narrower than the wing of the single winged vertical scotoma. The inner margin of this scotoma follows roughly the 15° circle, cutting the vertical meridian within the 20° circle and reaching the horizontal meridian near the 25° circle. The outer margin often passes almost directly upward, usually curving slightly toward the nasal side and cutting the vertical meridian near the 25° circle. The inner margin advances much more rapidly than the outer, until the nasal field is entirely filled in, leaving a small transversely oblong visual area on the temporal side between the blind spot and the fixation point. The temporal field is more slowly filled in by the steady advancement of the temporal margin. If one wing is more advanced than its fellow upon reaching the horizontal meridian on the nasal side, Roenne's step is formed.

The Single Winged Horizontal Scotoma.—This scotoma arises from the nasal side of the blind spot immediately above or below the horizontal meridian, and extends close to and parallel with this meridian until it passes the fixation point. After passing the fixation point it usually touches the horizontal meridian. As it develops it gradually covers the half of the visual field in which it lies, whether upper or lower, the

nasal quadrant being filled in first, then the temporal, ultimately producing a vertical hemianopsia. It is this type of scotoma that undoubtedly represented the Central Scotoma of Glaucoma mentioned by Elliot.[1] The remaining half of the field of vision may be invaded either by the development of another wing, horizontal or vertical, or by the advancement of the first wing beyond the horizontal meridian on the nasal side.

The Double Winged Horizontal Scotoma.—I have never seen this scotoma in its early stage, but I have examined a number of advanced fields in which there was left only a very narrow horizontal slit that must have developed in this way.

The glaucoma scotoma becomes larger with increased intraocular tension, and may become smaller, or even disappear, when this is diminished. It varies with the size of the test object used, and with the intensity of the illumination. While the double winged vertical scotoma usually advances symmetrically, ending with a very small central visual area, the single winged scotomata often produce fields that are varied and fantastic. It is probable that other factors than injury of the optic nerve fibers at the disk margin have a part in the production of these fields.

The observations here given only cover a period of about six years; they are, therefore, not final, and are not offered as representing a finished investigation, but only in the hope that they may stimulate in America an interest in this important subject.

---

[1] *A Treatise on Glaucoma*, Elliot, p. 314.

ON THE SUBJECTIVE CYCLOPIC EYE (THE THIRD EYE).

By Dr. SH. FUNAISHI.

(From the Department of Ophthalmology, Manchuria Medical College, Mukden.)

THE theory of an imaginary cyclopic eye first postulated by Hering and Helmholtz, and said by them to be midway between both eyes as the center of visual direction, has long been accepted generally.

My study, however, indicates that such a center of visual direction is not situated exactly between both eyes but in the head articulation (articulation of occipital bone with vertebral column) and that the center of monocular vision is also located there. The egocentric direction of an object is constant in binocular or monocular, in direct or indirect vision and the center of these directions is invariable even when turning the face in various directions so long as the body is fixed. In other words, the visual center depends merely upon the head articulation, and the manner of looking, therefore, has nothing to do with the visual direction and its center.

Concerning the cyclopic eye, I noticed, on various occasions, the following phenomena from which I have started to study this problem. I shall begin with the phenomena which may easily be proved by anybody.

Phenomenon I.

Closing the left eye, hold a pencil horizontally in front of the face at a certain distance (for instance at 30*cm*) and move it as if its end were directed towards the closed eye. By open-

ing the left eye, one will find that the pencil is pointing not at the left eye, but at the bridge of the nose. Thus, if the pencil is moved along its present direction it will touch the bridge of the nose a little beneath the level of the eyes.

Phenomenon II.

Look at an object at some distance in front with one eye while the other, say the left, is closed, then hold a pencil close to the seeing eye so as to cover the object with its end. One will then feel the end of the pencil to have moved to the right and upwards from the object as soon as he opens his left eye. At the same time, he will feel his right eye to have been located medially and lower than its first position.

These phenomena suggest that we optically and subjectively alter the point of vision of our own eyes, or that we have subjectively the positions of both eyes different from the true anatomical ones. I have carried out the following experiments in order to determine the altered subjective position of the eyes.

Experiment I.

a. The head of a man is fixed by means of a forehead and chin-rest at one edge of a table and a row of small paraffin balls is held over the other edge at the height of the eyes. The subject looking at the paraffin balls with one eye closed, marks with pencil on paper on the table several points along the directions from the closed eye to each paraffin ball. The extension of those lines drawn through the points along each direction pass through a small area situated about 10*cm* behind the vertical plane of the eyes in media.

b. The table in the above experiment is excluded from the sight and only the row of paraffin balls is shown and the lines of directions of the seeing eye to each paraffin ball is pointed out with the finger on measuring tape previously placed on the table. The lines of directions of the seeing eye from each paraffin ball converge into the same area as before.

c. One shows in a dark room the line of direction from the eye to a point of light instead of to a paraffin ball by a simi-

lar arrangement, pointing out the direction on the measuring tape with a finger, he then lifts the finger and again points out the direction of the other eye to the same light. The lines of direction of both eyes from the point of light is just the same.

d. In the same way, further experiments were made with a vertical row of balls and a vertical plate. The converging area is then found about 10*cm* behind the eyes and about 4.5*cm* beneath them in the head articulation.

Experiment II.

a. If one looks at an object at a certain distance in front of a closed eye and points out the middle point between the closed eye and the object, the point stands much nearer to the eye than to the object. Indeed it stands about half way between the object and the external opening of his ear.

b. Let one look at an object with one eye through a tube which excludes the greater part of the visual field, and point out the middle point between the seeing eye and the object. The result is same as before.

Here, too, we feel as if our seeing eye as well as the closed eye has been moved about 10*cm* behind its true position.

Experiment III.

We can also prove directly the subjective position of our own eyes.

a. If a closed eye, or a seeing eye through a tube, is pointed with a finger from the side, the finger will direct slightly behind and below the opening of the ear.

b. If a pencil be held horizontally with both hands behind the head at the height of both eyes, it practically stands just at the height of the head articulation.

c. If a pencil be held horizontally over the head so that it seems to lie in the vertical plane of the closed eye, it lies, in fact, above the head articulation.

d. Repeat the experiment in Phenomenon I, it will be found that a position between the eyes is pointed to.

Experiment IV.

A phenomenon similar to that of my Phenomenon II, has been observed by Wessely in 1913. He threw the light on a closed eye from various directions through the lids, and found that the subject always felt as if the light were situated temporally. This phenomenon was later studied by Dimmer, Kollner, Shoen, etc., and lately by Landolt. I also made the same experiment and found that the light subjectively projected upwards and towards the temporal, just as in my Phenomenon II. Attention should be paid to the fact that the light is felt to penetrate into the face to the depth of the head articulation.

We feel our own eyes to lie in the head articulation and the eyelids laterally and upwards from the situation of the subjective eyes. Therefore, it is no wonder that the light thrown upon the lids is projected in the direction of the lids from the subjective eyes.

Conclusion.

I. We have two organic eyes but transpose them optically and subjectively at the head articulation (articulation of occipital bone with vertebral column) where the center of visual direction lies.

II. We find no difference between the eyes thus transposed in actual vision. Hence we have only one subjective eye which may be called "the subjective cyclopic eye or the third eye."

BIBLIOGRAPHY.

Funaishi. *Graefe's Archiv für Ophthalmologie.* Vol. 116, p. 126.
Wessely. *Klinische Monatsblätter für Augenheilkunde.* Vol. 51, p. 596.
Köllner. *Archiv für Augenheilkunde.* Vol. 76, p. 153.
Dimmer. *Graefe's Archiv für Ophthalmologie.* Vol. 105, p. 794.
Landolt. *American Journal of Ophthalmology.* Vol. 7, p. 595.

A CASE OF TEAR INTO THE CILIARY BODY FROM CONTUSION.

By Dr. H. D. LAMB, St. Louis, Mo.

(With two illustrations on Text-Plate XIII.)

R. E., 9 years old, was struck in the right eye by the hanging loop of a swinging rope; this was immediately followed by hyphæmia to a height of *2mm* and a moderate iritis; forty-eight hours after the accident there was some pain in the eye but the appearance of the eye was unchanged, chamber deep, pupil dilated about halfway with atropine. Three and one-half days after the accident there was considerable pain in the eye and the anterior chamber filled with blood; tension only a little elevated to fingers, perception of light good but projection poor. In spite of miotics, pain continued with no change in the hyphæmia. Tension was never more than a little elevated. Consultations were held with Dr. A. E. Ewing and Dr. N. R. Donnell.

Three weeks after the accident the eye was enucleated because of the uninterrupted pain, no improvement in vision and no sign of any absorption of the blood from the anterior chamber; near superior and inferior limbi there was present a small area showing a dark discoloration.

The eyeball was fixed in Zenker's solution.

Macroscopic.—The sclera is distinctly thinner than normal over the equatorial region. The anterior chamber is filled with blood. The lens is thin and disc-shaped except for a shallow indentation in the middle of its anterior surface. The optic nerve shows a well-marked physiological cup. Detachments of the choroid and retina are artefacts.

The eyeball was dehydrated and imbedded in celloidin; the sections were stained in hemotoxylin and eosin stains. On sectioning, it was discovered that in the dehydrating solutions the cornea had become flattened laterally, the cornea bulging anteriorly and the anterior chamber very deep.

ILLUSTRATING DR. H. D. LAMB'S ARTICLE, "A CASE OF TEAR INTO THE CILIARY BODY FROM CONTUSION."

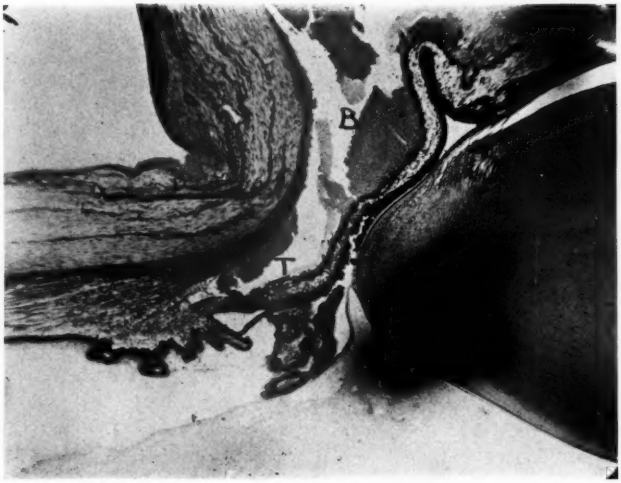


FIG. 1. Section under medium power through the iris angle on the nasal side showing, T, tear into ciliary body, B, blood.

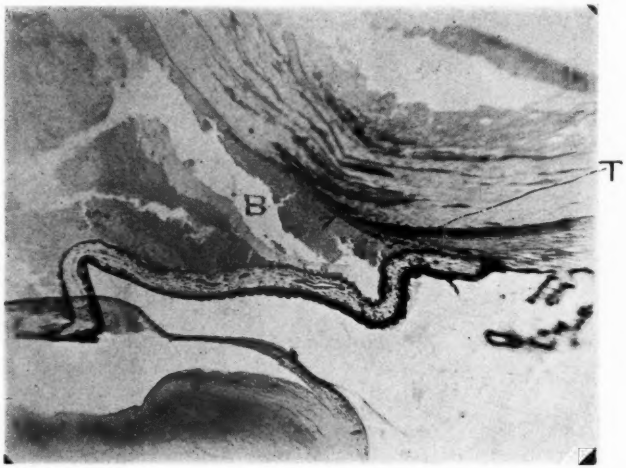


FIG. 2. Section under medium power through the iris angle on the temporal side, showing, T, tear into ciliary body, B, blood.

UNIV. OF MICH.

Microscopic.—The cornea shows considerable desquamation of the anterior epithelium. Many capillaries are seen in the epithelial layers. Many capillaries are seen in the extreme peripheral parts of the cornea, with streaks of hemorrhage showing in some places between the corneal lamellæ.

The trabeculæ of the filtration net-work contain red corpuscles.

The anterior chamber is almost completely filled with blood; some of this blood posteriorly has broken down into a granular detritus.

The iris in its entire thickness is in places folded or wrinkled. The iris is atrophic, being about one-half the normal thickness. There is a moderate proliferation of the chromatophores, mostly of the unpigmented ones. The blood vessels are reduced to thin-walled tubes, to capillaries for the most part. There are fewer muscle cells and fibers of the sphincter pupillæ muscle; an increased amount of albumen is found in the fluid in the stroma of the iris. The posterior pigmented epithelium shows a thickening in some places, loss of pigment in other places, a thinning or breaking up of the epithelium in still other spots; posterior synechiæ are present in a few places.

The iris and ciliary body show the essential changes that resulted in the loss of the eye. This change is principally a meridional or longitudinal tear or splitting through the trabeculæ of the ligamentum pectinatum at the base of the iris and extending posteriorly and internally just inside the longitudinal and oblique bundles of the ciliary muscle, thus causing the anterior chamber at the iris-angle to extend posteriorly farther than normal. The depth of this tear is quite uniform around the circumference of the eye, varying between 1½ and *2mm.* The separated internal strip of tissue is about the same thickness as the iris; it is lined almost throughout its extent by two layers of pigment iris-epithelium; a cystic separation between these pigment epithelial layers is seen at the extreme outer end of the iris in some places. Following the rent in the tissues there has been a falling backwards and inwards of the base of the iris. The appearance is given under the microscope of the iris being wider from pupil to base than is normal. On the nasal side, the scleral spur to which the longitudinal bundles of the ciliary muscle are attached, is seen to be far anterior to the bottom of the tear or farthest extent of the new iris-angle. On the temporal side of the eye the inner separated strip in its posterior portion has later come in apposition with the adjoining denuded surface of the ciliary body. It is evident that there has also been a longi-

tudinal contraction or drawing together of the tissues in the separated strip. Much hemorrhage is present in the gap as also inside of the adjoining tissue. There are a few small lymphocytes in the tissue of the ciliary body adjoining the tear on the nasal side; on neither side are there seen signs of the rent attempting to heal up.

The ciliary body is atrophic; there is a diminution of the ciliary muscle with no accompanying sclerosis. There are present in the vascular layer many new-formed capillaries, containing polymorphonuclear leucocytes. In addition are seen here many plasma cells and a proliferation of chromatophores. Some cystic degeneration is seen in elongated cells of the unpigmented ciliary epithelium in the posterior part of the orbiculus ciliaris.

The lens shows loss of the anterior epithelium in an area corresponding to the dilated pupillary area and a liquefaction of the superficial lens fibers in a very narrow strip adjoining the lens-capsule over the same area. Beyond the pupillary area on each side there is a layer of superficial cortical lens fibers containing small vacuoles; this layer increases gradually in thickness as the equator is reached, where it is the thickest; it ends just behind the equator. The indentation in the anterior portion of the lens can be explained only by the unequal shrinking of the lens in the dehydrating fluids. The choroid is atrophic particularly in its anterior and middle portions; there are present many new formed capillaries and a proliferation of the chromatophores.

The pigment retinal epithelium shows loss of pigment.

The retina in its anterior half shows a cystic degeneration frequently quite coarse in the nerve fiber layer. A fine cystic degeneration is present in much of the ganglion cell layer and in the outer part of the rods and cones. Posteriorly there is seen a marked proliferation of neuroglia cells.

The optic nerve shows no change.

Summary and Conclusions.

It is difficult to explain the late onset of violent changes in this case; it will be remembered that it was not until three and one-half days after the trauma that the most marked hemorrhage occurred. Perhaps the ciliary arteries at the junction of the iris and ciliary body were torn across in such a way as to first block off the outflow of blood into the anterior chamber. Possibly later with increased congestion and

softening of the clot and torn vessel walls, the hemorrhage was renewed in an intense degree.

The atrophy of the iris, ciliary body and anterior choroid would naturally follow the tear through the base of the iris into the ciliary body, not only because of the interference with the circulation but also because of the division of trophic nerves.

The changes in the lens are interesting. The destruction of the anterior epithelium and just adjoining cortical fibers are probably directly due to the blow on the eye. The cataract changes near the equator of the lens are more likely due to chemical changes in the surrounding aqueous dependent upon injury to the ciliary body.

The tear in the ciliary body would naturally be expected to resemble a tear in the iris in not healing or showing any tendency to close up the rent.

There are very few similar cases in the literature.

Wagenmann (1) reports an iridodialysis resulting from a contusion on the ball, the eye was enucleated one year after the injury. The iris was found roughly torn at its ciliary attachment. The tear showing no cicatricial tissue was covered only by an endothelial layer. Schäfer (2) had found the tear stopped by the pigment epithelium of the iris. The ligamentum pectinatum in the vicinity of the tear appears torn through roughly, the iris appearing behind the scleral spur. The loosened part of the iris is much shrunken, sclerosed and irregularly pigmented; the iris-vessels are obliterated. The corresponding part of the ciliary body is atrophic. Corresponding to the iris-tear, there was a tear in the zonular fibers. Latterly from the iridodyalysis in the entire circumference of the ball, there was a tear into the chamber angle, splitting of the ligamentum with the formation of a cleft into the tissue of the ciliary body. In one part the fissure gaped, in another part the superficial part of the iris was united by a few fibers with the ligamentum.

Teich (3) reported an essentially similar case. The eye was enucleated three weeks after the injury. In places the iris was completely torn away at the ciliary body, in places there was still a stump. Here there was also an endothelial overgrowth. The tear in the ligamentum extended further and

opened Schlemm's canal. The zonular fibers were torn in a linear streak and the lens displaced. There occurred thereby a detachment of the ciliary body and the adjoining part of the choroid by the filtrating or directly flowing off aqueous humor.

1. WAGENMANN. "Injuries to the Eye." *Graefe-Saemisch, Handbuch der Augenheilkunde*, 2 Auflage, IX Band, 5 Abt., p. 4.
2. SCHÄFER. *Archiv f. Ophth., 1883.* XXIX, 1, p. 13.
3. TEICH. *Archiv f. Augenheilk.*, 1905. LII, p. 261.

A SIMPLIFIED CONCEPTION OF VISUAL FIELD CHANGES IN CHRONIC GLAUCOMA.[1]

By Dr. LUTHER C. PETER, Philadelphia.

THE outstanding characteristics of visual field changes in chronic glaucoma are enlargements of Mariotte's blind spot above or below, or in both directions—a symptom known as Seidel's sign; a sickle-shaped scotoma of varying modifications—a Bjerrum scotoma; peripheral loss on the nasal side, often a step-like formation called Roenne's step; in the late stages, concentric contraction even to a small island five degrees around the fixation point, with or without a small detached temporal area of vision; complete central and peripheral loss with the exception of a small peripheral detached area in the temporal field; preservation of color sensitivity in visual areas; an occasional temporal quadrantic defect and peripheral contraction which seems to be more or less concentric.

Most of these field changes have been carefully analyzed and to each has been assigned a special name, such as Seidel's sign, Bjerrum scotoma, Roenne's step, Elliot's sign, etc. Less attention has been paid to the gradual peripheral contraction of the entire field and to the occasional temporal sector defect, largely because they are of infrequent occurrence. The four special signs or symptoms, to which definite names have been given as a tribute to those who especially studied and developed the defect in the field, are examples of the same process, in which, however, different groups or bundles of retinal nerve fibers suffer from increased intra-ocular tension. The preservation of color sensitivity in visual areas is further

[1] Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, December, 1926.

evidence of this same selective group or bundle atrophy, with which we are familiar.

Peripheral contraction and temporal quadrantic defects require more searching analysis to harmonize them with what otherwise seems to be a uniform process of field pathology.

According to Collins and Mayou (*Pathology and Bacteriology of the Eye*, p. 318) "the disturbance of the function of the retina, as the result of increased tension, may be due either to diminished blood supply or to atrophy of its nerve elements. Loss of vision due solely to the first cause is recoverable; that due to the second is permanent." The same authors further state, "in eyes blinded by glaucoma, atrophy of the nerve elements of the retina occurs, *the ganglion cells become vacuolated and after a time disappear:* the outer limbs of the rods and cones become flattened by intra-ocular tension against the pigment cell layer, and after a time become much bent, *but do not atrophy*." They further believe that "failure of the field of vision which occurs in chronic glaucoma, though it may, to some extent, be nutritional in origin, is certainly largely due to damage to the optic nerve fibers from stretching and pressure." In this concise statement of facts, in which most ophthalmic surgeons concur, we find the explanation of the Bjerrum scotoma and all its modifications. It is not essential, for the purpose of this communication, to go into details to explain just why these special defects occur in glaucoma, but in order to further analyze peripheral concentric contraction and temporal quadrantic defects as they occasionally occur, some of the reasons for the selective type of field changes should be discussed.

The general conception of the arrangement and distribution of the retinal nerve fibers is that suggested by Fuchs. Fibers which pass to the nasal retina radiate like the spokes of a wheel. Those to the temporal side assume an arcuate direction, passing above and below the direct macular fibers and terminate at the raphe. The long fibers which terminate in the peripheral retina probably occupy the central area of the optic disk. Those which terminate intermediately, pass into the nerve head midway between the center and the periphery of the nerve. Short fibers, which pass to the retina contiguous to or immediately surrounding the nerve, occupy the

most peripheral part of the nerve. The presence of the cup on the temporal side of the disk accounts for the usual early appearance of a Bjerrum defect in the nasal field. Such defect may be caused primarily by a stretching process of the long peripheral temporal fibers, or by a cutting-off of the nutrition of short fibers by their contact with the hard scleral ring, as the result of increased intra-ocular pressure. One may, therefore, find the first evidence of field pathology in the form (a) of a Roenne step, (b) a typical Bjerrum scotoma, (c) a Seidel or Elliot sign. In the formation of any one of these symptoms, the same process is in evidence—namely, a blocking off of a group or bundle of nerve fibers which are distributed to a definite area. In other words, each of these defects is in fact a sector defect, or a Bjerrum scotoma, regardless of the part of the nasal field in which it may occur. That our conception of the process should be clouded and confused by the multiplication of names is unfortunate. The contributions of Roenne, Seidel, and especially of Elliot, to the subject of glaucoma will not be dimmed by regarding the sector-like defect, from which the entire glaucomatic field evolves, as a Bjerrum scotoma.

In the occasional instances in which the nasal retina suffers first as shown by a superior or inferior temporal cutting, is the defect similar to that observed in the usual Bjerrum nasal scotoma? It probably is of the same character. Although the nasal fibers as a rule suffer late, it is possible that they may be the first to yield to the stretching and pressure process. If atrophy of the temporal fibers were due solely to pressure against the sharp scleral edge on the temporal side of the cup, it is probable that the first fibers to be blocked would be the short fibers just beyond the disk, and the Bjerrum scotoma would begin above or below the disk and continue outward. It is a well-known fact, however, that this defect may begin as an extension of Mariotte's blind spot, in a detached area above or below the point of fixation, or in the extreme periphery of the nasal field, with a typical step-like formation. It is probable, therefore, that those fibers which are more centrally located in the optic nerve are cut off by a stretching process. While the stretching usually reaches the temporal fibers first, it is possible that the long nasal fibers

may suffer at the same time, or even before the temporal fibers yield. The direction of entrance of the optic nerve, as it passes into the sclera, and the direction of the development of the cup, can easily account for this variation or digression from the usual course of developments. In fact, these same conditions readily account for the occasional tendency to general peripheral loss before central defects appear.

That peripheral contraction is not due to pressure upon the chorio-capillaris, as claimed by some, can be fairly well established by the fact that the rods and cones are not found to be atrophic, but only distorted. If their nutrition from the chorio-capillaris were to fail, the atrophic process would begin in the rods and cones. As pointed out by Collins and Mayou, these receptive end organs are not atrophied. Peripheral field loss through failure of the central retinal circulation is less doubtful in chronic glaucoma than that of the choroidal circulation. In fact, although not the primary factor, it is a contributing cause. Elliot has appropriately pointed out, in his classic on Glaucoma, that marked peripheral concentric contraction may be induced by other causes operative in conjunction with glaucoma. Admitting this thought of Elliot, as a cause for peripheral contraction, and the possible contribution of nutritional disturbance in the peripheral retinal circulation, the stretching process of the long fibers, whether in the nasal or temporal field, may extend to all parts of the periphery. It is not difficult, therefore, to harmonize general peripheral loss with the other more characteristic phenomena, aided, perhaps, in part by nutritional disturbance of the circulation which is derived from the branches of the central retinal artery.

Preservation of color sensitivity has been cited as characteristic in chronic glaucoma. This statement, however, needs careful interpretation. It is quite true that color behavior differs in the atrophy of glaucoma from that ordinarily observed in other forms of atrophy. In parts of the field which have not suffered from loss of form, or in which nerve fibers have not been subjected to the stretching or pressure process, color fields are preserved. On the other hand, in sectors which begin to show evidence of invasion, color loss, especially that for green, is the earliest sign of the advancing process.

For example, when a typical Bjerrum scotoma exists above the point of fixation, a careful search below this point between the tenth and twentieth degree, by a green test object, may uncover an area insensitive to green, when even the smallest white stimulus will register insensitivity or fail to uncover a defect. The behavior of colors, therefore, especially of red and green, in the distribution of fiber groups which are yielding to the glaucomatic process, does not differ from that which is observed throughout the field in other types of atrophy. Preservation of color sensitivity in small concentric islands surrounding the macula is but evidence that the nerve fibers supplying these areas have not been subjected to the pressure or stretching process. Detached areas in the temporal fields, although definitely sensitive to form and light are indifferently responsive to colors. These areas, although preserved to form, are damaged areas at best as shown by indifferent and uncertain response to colored stimuli. It is this special response to color, as well as to form, that gives character to the atrophy of glaucoma in contrast to other forms of atrophy. It marks the beginning of atrophy of definite groups of nerve fibers.

This selective process is in evidence in every phase of visual field changes observed in glaucoma. It applies to temporal defects as well as to the more usual nasal sectors; to the periphery of the retina as well as to the occasional case in which the macular fibers are the first to suffer. The frequency of one form of field defect and the infrequency of another does not change the character of the process. The order in which these various changes appear, is determined by difference in the anatomic relation of the nerve head to the sclera, the strength or weakness of different areas in the cribriform membrane, the supporting fibers of the nerve, and the contingent development of the glaucomatic cup. The underlying process is always the same. In the interests of simplicity, therefore, and a better conception of the various changes which one encounters in the field pathology of glaucoma, the Bjerrum scotoma or sector defect may be regarded as the basic principle which will explain any phase of these field changes. It was Bjerrum who first called attention to the peculiar arched sector-like

defect which is now known to be the characteristic form of field change in glaucoma. Tracing these defects back to the pressure and stretching process as a cause, it becomes quite apparent that the part of the field which will first be invaded is determined by the anatomic relation to which we have referred. Furthermore, the subsequent evolution of the field pathology in any given case is determined by the same factors.

Changes observed in acute inflammatory glaucoma have intentionally been omitted. In this condition, nutritional disturbances largely predominate, although permanent defects are brought about by the same causes which operate in chronic glaucoma.

It is often characteristic in glaucoma simplex, to find intra-ocular tension easily within normal limits upon repeated examinations, while the fields progressively contract in the usual characteristic manner. Inability to detect increased intra-ocular tension can not be construed as evidence that hyper-tension does not exist. As Elliot has observed, we know little of the state of tension at night, when the cardio-vascular system is at its lowest ebb. Veinous stasis may be sufficient under these conditions to raise the intra-ocular tension to a level which will maintain the stretching process. By some this type of glaucoma is thought to differ from other forms of chronic glaucoma. Field studies, however, are the same and it is likely that increased tension is sufficient at times to keep up the process in eyes which either are essentially weak and non-resisting, or in eyes which have lost their resistance after many years of an insidiously developing process. To the writer, this last group of cases seems to follow the same laws as do other forms of chronic glaucoma, modified only by the greater length of time during which they have been subjected to the stretching and pressure process.

This simplified conception of the field changes in chronic glaucoma is best summarized in the words of Elliot: "The great underlying factor in chronic glaucoma is an interference with the nerve bundles, either on the optic disk or as they emerge from it. Other factors are only contributory." (*Treatise on Glaucoma*, Second Edition, page 306.)

If this thought is constantly kept in mind, the ofttimes bizarre and unusual fields uncovered in glaucoma, as well as those forms which are regarded as the usual types, are reduced to a basis of uniform simplicity.

DIVERGENCE EXCESS: ITS DIAGNOSIS AND TREATMENT.

By Dr. JOHN H. DUNNINGTON, New York.

TO properly understand the condition of divergence excess we must first consider what produces the act of divergence. Some writers still maintain that divergence is a purely passive affair due entirely to a relaxation of convergence. Duane and other authorities have, for a long time, agreed that divergence is an active as well as a passive process being brought about by a simultaneous contraction of both external recti associated with the relaxation of the internal recti. When our eyes are directed at a near object the power of divergence is in a state of relaxation and the converging power is stimulated, whereas on looking at an object twenty feet or more away there is a complete relaxation of the power of convergence and an active stimulation of the center of divergence. The power of divergence is used to maintain parallelism when we look at distant objects.

Diagnosis of Divergence Excess.

A pure case of divergence excess presents the following findings:

1.—Marked exophoria for distance.
2.—Excessive prism divergence (abduction).
3.—Normal prism convergence (adduction).
4.—Normal muscular balance at thirteen inches.
5.—Normal near point of convergence.
6.—Lateral movements of each eye inward and outward normal in extent and comitant.
7.—Diplopia, if present, not increasing to right or to left.

I will now consider these signs in detail.

1. *Exophoria for Distance.*—There is a marked exophoria at twenty feet, *i.e.*, from 5 to 30 or more degrees. A case can be just as true a divergence excess with 5° of exophoria as one with 40°. The most commonly used instruments for determining the amount of exophoria present are the Maddox rod and the phorometer. I prefer in this class of cases to use the screen and parallax test. With this test there is absolutely no tendency for the patient to converge and thereby overcome a part of the deviation. When the Maddox rod is used the patient makes an effort to line up the streak and light in this way diminishing the amount of exophoria. I have seen a difference of 15° in the amount of exophoria as shown by the Maddox rod and by the screen and parallax. In divergence excess the Maddox rod invariably shows a smaller amount of exophoria. The same fault can be found with the phorometer only to a lesser degree. The screen and parallax test shows the actual amount of outward deviation present when the eyes are at rest. In those cases with an actual divergent squint the screen and parallax test is the only test we have, to accurately estimate the amount of divergence present. The screen and parallax test is therefore the text *par excellence* to determine the amount of exophoria or exotropia caused by a divergence excess.

2. *Prism Divergence.*—The power of overcoming a prism placed base in, the so-called prism divergence–(abduction), is an absolute definite thing. It does not vary from day to day nor does it improve with exercise. The normal limits of this power as set by Duane are from 3° to 8° when measured at twenty feet. An ability to overcome a prism placed base in of more than 8° denotes an excessive power of prism divergence. This increased prism divergence is a constant finding in divergence excess. The actual amount these patients can overcome will depend upon the degree of excess of divergence present. The highest prism divergence I have seen was 33°. It usually corresponds fairly closely to the amount of exophoria as found with the screen and parallax test. This increased prism divergence and high degree of exophoria at twenty feet are pathognomonic of divergence excess.

3. *Prism Convergence.*—The prism convergence (adduction), for twenty feet is normal. This ability to overcome

prisms placed base out is not constant like the prism divergence. Since it does vary our normal limits must be more elastic. They are usually quoted as ranging from 15° to 30°. In divergence excess this power of prism convergence should be within these figures. Only in those cases where there is a secondary weakening of the power of convergence would it be below normal.

4. *Muscular Balance at Thirteen Inches.*—The amount of deviation present at thirteen inches should be within the normal limits, *i.e.*, it would range anywhere from orthophoria to 4° of exophoria. Here again I prefer the screen and parallax test over the Maddox rod or phorometer for the same reason as given above. Any abnormality of the findings at this distance denotes an anomaly of the power of convergence.

5. *Near Point of Convergence.*—A normal near point of convergence is roughly one not more than 90*mm* from the center of rotation of the eyeballs. This is usually estimated with a 3*mm* white headed pin or a 2*mm* black dot on a white card. I prefer a small light such as a May ophthalmoscope with the cap removed. With this as a test object I believe the ordinary patient will maintain more accurate fixation. The near point of convergence should be normal in divergence excess. Any persistent remoteness of it means a convergence insufficiency, as this is the most accurate test we have to determine the maximum power of convergence.

6. *Lateral Movements of the Eyes.*—There will not be any limitation of motility nor any overaction of either eye in any of the cardinal directions of gaze. This affection is not an individual muscle trouble therefore the relation of the two visual axes will not change in the different directions of gaze. That is if there is 10° of divergence in the primary position there will be 10° in all fields. When a divergence excess has become sufficiently marked to produce a constant divergent strabismus we do get secondary muscular changes.

7. *Diplopia.*—Diplopia is not a constant finding but occasionally it is a very troublesome feature. A double vision due to divergence excess increases as the object is carried away from the eyes and diminishes as the eyes are approached. Frequently in these cases there is diplopia only at a distance of twenty feet or more with binocular single vision at any closer

range. The diplopia when present is crossed and does not vary in amount in either lateral field. It does increase as you go above the horizontal plane and becomes less in the lower fields. This is true of all crossed diplopias. It has been explained by Duane as being due to the natural tendency for us to converge our eyes on looking down and to diverge them on looking up. An homonymous diplopia behaves in an exactly opposite manner. The characteristic feature then of a diplopia caused by an excess of divergence is its variation in amount as the distance between the test object and the patient changes.

Differential Diagnosis.

The two chief conditions to be confused with divergence excess are convergence insufficiency and insufficiency of an adductor.

Convergence insufficiency shows normal distance findings, marked exophoria at thirteen inches and a remote near point of convergence. As we have seen an excess of divergence gives normal near findings and abnormal distance ones. The differentiation lies in doing muscle tests for distance and for near. Consideration of which is pathological will tell you the etiology of the exophoria present. If an exophoria is greater in amount at twenty feet than at thirteen inches it is primarily due to a divergence excess. If the exophoria at near range is more marked it is caused by a convergence insufficiency.

Insufficiency of an Adductor.—From an insufficiency of an adductor (internal rectus, superior and inferior recti) divergence excess is differentiated by the fact that in the former condition either the power of movement of the affected eye inward is obviously impaired or at all events the exophoria and the crossed diplopia increase definitely as the eyes are carried to one side or the other.

In many cases a divergence excess becomes complicated with a secondary convergence insufficiency or vice versa. When the divergence excess is the primary condition we have a marked exophoria for distance with a smaller amount of exophoria for near. Frequently in such cases there will be a divergence squint for distance and only an exophoria for near.

Etiology.

Divergence excess is either primary or secondary.

Primary Divergence Excess.—The exact etiology of this condition is not thoroughly understood. It is found in childhood or early life in its uncomplicated form. Later on in life there is a gradual secondary weakening of convergence to complicate the picture.

As Wootton pointed out in 1915 divergence excess is more commonly found in hyperopes than in myopes. This I believe to be mere coincidence due to the larger number of hyperopes seen. It is difficult for me to imagine that any refractive error could produce this condition in its primary form. It must be the result of an inherent overactivity of the center of divergence, hypothetical as that may be.

Occasionally primary divergence excess is the cause of an actual divergent strabismus in the first few years of life. However most of these cases can overcome the deviation and not until the age of puberty is reached does it just make itself known. Once in a while we see a patient with a primary divergence excess come complaining of a momentary convergent strabismus. This transitory turning in of the eyes is due to a spasm of convergence which nature has brought about in her effort to check the outward deviation.

Secondary Divergence Excess.—This condition follows a primary convergence insufficiency. It is not possible at this time to consider the causes of the weakened power of convergence. Any insufficiency of convergence if neglected and allowed to progress will in time become complicated with a divergence excess.

Symptoms.

Divergence excess may be present for years without causing any symptoms. The majority of these cases, however, experience difficulty in overcoming this exophoria. They have headache and pronounced asthenopic symptoms (feeling of strain and tiring in the eyes) together with a sense of confusion on watching distant objects, particularly when moving or brightly illuminated. For this train of symptoms Bennett

as far back as 1897 coined in the term panoramic asthenopia. These patients are particularly liable to be troubled with car sickness. They usually abhor crowds or moving objects of any kind. Some cases are not aware of any discomfort at a distance but complain of inability to do long sustained near work. These patients will always show a secondary weakening of the power of convergence. Their symptoms are then due to the convergence insufficiency which has been caused by the divergence excess. In still others the gastric symptoms predominate.

Course.

A primary divergence excess may remain stationary for years, however, the tendency is for the condition to slowly progress. If these cases are neglected the exophoria for distance gradually becomes more and more marked until the patient is no longer able to overcome the tendency of his eyes to deviate outward and he develops an actual divergent strabismus for distance. Accompanying this change in the distance findings is a gradual progressive weakening of the power of convergence until finally a constant divergent strabismus for both distance and near result. In those cases where the convergence insufficiency is primary and the divergence excess secondary the order is just reversed. These patients have first a divergent squint for near then later one for distance. In either case the final result is the same—a concomitant divergent strabismus.

Treatment.

Prismatic correction of an exophoria caused by a divergence excess is not satisfactory as small amounts of exophoria (less than 5°) of this etiology do not usually cause symptoms. Any deviation large enough to cause annoying symptoms is too great to be corrected by the use of prisms. These prisms would furthermore be needed only for distant wear and if used constantly would produce a secondary convergence insufficiency.

The treatment of primary divergence excess is purely operative. It is the only way to permanently cure the condition.

The operation of choice is a complete tenotomy of the external rectus of one or both eyes depending upon the amount of deviation present. I personally perform an open tenotomy of one external rectus and if an over-correction of 10 or more prism degrees is not produced I do the same procedure on the other eye. I operate first on the eye showing the greatest tendency to deviate. In exophoria or exotropia of twenty or more prism degrees at twenty feet I believe you can safely do a complete tenotomy of each external rectus at one sitting. However there is no disadvantage in operating on only one side at a time. An immediate over-correction of approximately fifteen prism degrees is in my opinion about ideal. Should a bilateral complete tenotomy of the external recti leave the patient with an under-correction I should advise waiting a month and then repeating the procedure. I see no reason for shortening the internal recti as long as the power of convergence is good.

In those cases where there is a secondary convergence insufficiency in which their symptoms are most pronounced on close use of their eyes, improving the power of convergence by exercise will give them temporary relief. However, unless the primary divergence excess is removed by operative means there will be a return of the symptoms. The ideal treatment here is first to correct the divergence excess and then use converging exercises. In secondary divergence excess which follows primary convergence insufficiency we first correct by exercise or other means the weakened converging power and later if necessary tenotomize to remove the divergence excess. Here our primary cause is the convergence insufficiency so our treatment should first be directed at it.

Summary.

1. Divergence excess is a common cause of exophoria or exotropia.

2. The diagnosis of it is made by a comparison of the muscular imbalance at 20 feet and at 13 inches along with an estimation of the near point of convergence.

3. It is not refractive in origin and can only be cured by surgical means.

A SIMPLE APPARATUS FOR DETERMINING THE NEAR POINT OF ACCOMMODATION AND THE ASTIGMATIC AXIS.[1]

By Dr. T. P. LEE.

(Department of Ophthalmology, Peking Union Medical College, Peking, China.)

(*With two illustrations in the text.*)

THE results of tests for the range of accommodation vary greatly, as is pointed out by Howard (1) in his presentation of a new apparatus for testing the accommodation. Among the factors causing the differences, he mentions the following (1) non-uniformity in the size of the test objects; (2) differing illumination of the test objects; (3) varying degrees of cooperation on the part of the subjects; and (4) the personal equation of both the subjects and the examiners. The test becomes even more complicated when uncorrected errors of refraction exist; in fact, the results obtained by the usual methods in cases of uncorrected astigmatism are so uncertain that the records of the range of accommodation of such cases might well be considered worthless. Theoretically and ideally, no test for accommodation should be made unless the subject has first been made emmetropic by placing in front of his eyes the full correction of his error, both spherical and cylindrical. Practically, it is impossible to do this in most cases, since they often come either without any correcting glasses or wearing glasses that obviously need changing following a cycloplegic test.

Even with Duane's (2) accommodation disc we have fre-

[1] Read before the Section on Ophthalmology of the China Medical Association at the Biennial Conference in Peking, September 1–8, 1926.

quently found the test for accommodation unsatisfactory, because patients with only a moderate degree of astigmatism sometimes cannot distinctly see the fine line on the disc at any distance. The apparatus is so constructed that the line is always set in a vertical position. We have found that when astigmatism is simple and has the axis of its correcting cylinder at 180°, the range of accommodation can be uniformly and accurately measured with Duane's apparatus without the correction on. For in this case the patient sees the vertical line of the test object clearly at the same distance from the eye as it does with the correcting cylinder on. But the result is uniform and accurate in uncorrected cases only under this condition, *i.e.*, in simple astigmatism with axis at 180°. In simple astigmatism with its cylindrical axis at 90° the vertical line on Duane's disc often cannot be seen clearly at any distance. For in this case a horizontal line is seen most distinctly. Theoretically, the vertical line should appear as a blurred band for all distances; practically, many patients see it more or less clearly, but at a subnormal distance. That is, there is recorded too low a range of accommodation for the patient's age. Generally, too, the distinctness varies from moment to moment, since most eyes evidently possess some power of adjustment for astigmatic errors. But on the whole, the test applied is usually unsatisfactory. Since the majority of cases that are astigmatic have their axes at some other position on the arc than at 90° or 180°, it is obvious that some other method than we have at present ought to be applied. That method must take cognizance of the existing astigmatism by providing an adjustment of the axis of the test line on the disc so that it may be set exactly at right angles to the astigmatic axis. The simple apparatus proposed below meets the essential requirements:

1. An accommodation disc set in a round metal frame. It seems preferable to use a small white square, 5*mm*, 5*mm*, set in the center of a circular black disc. Two very fine parallel lines, about half a millimeter apart on the white square, make an excellent test object—in fact, better than a single line. On the white back of the disc a heavy diameter line is drawn, parallel to the two lines on the white square, which is on the front of the disc (Fig. 1).

2. ***A rotating carriage.*** The accommodation disc is inserted in a rotating carrier controlled by a small ratchet on the side.

FIG. 1.

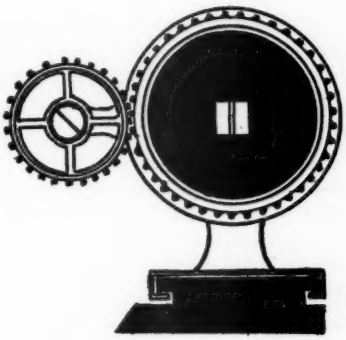
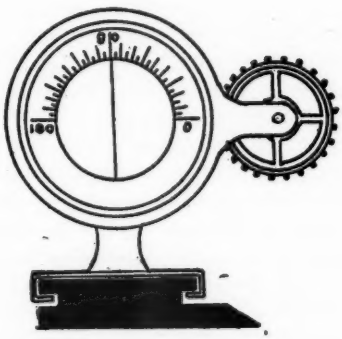


FIG. 2.



The carrier is held in a circular frame, on the posterior surface of which is inscribed a scale graduated in degrees. When the disc is turned in the carrier, the line on its back indicates the axis of the test lines on its front. The carrier with its frame is mounted on a metal base made to slide smoothly over a Prince's rule (Fig. 2).

3. Prince's accommodation rule. In applying the test with this simple apparatus, we first determine the axis of the test line at which maximum clarity is obtained by the patient. Then the test object may be brought slowly and steadily toward the eye until the first blurring of the test line is noticed. During the approximation of the accommodation disc toward the eye, it is well to have the patient wink frequently in order to prevent a spasm of accommodation.

Since we test the accommodation before giving a cycloplegic in our routine examination, and since we are dealing with uncorrected errors in most of our cases, one of two conditions will obtain when applying the test:

a. The test object may appear equally clear in all positions of the arc, in which case it is evident that there is no astigmatism only a very insignificant degree of it in the eye under examination. The figure obtained for the near point can then be made to correspond to that finding when the eye in question is made emmetropic by simply adding or subtracting, respectively, the amount of hypermetropia or myopia which has been found following the use of a cycloplegic.

b. The test object may appear most distinct at one position. This proves that astigmatism is present and in a sufficient degree to be considered. From the position of the test object, also, the axis of the existing astigmatism is thereby known, *i.e.*, at right angles to that position at which maximum clarity of the test object is obtained.

This simple apparatus contains no new principles but is simply a new combination of two or three already well-known principles. The test disc in a rotating carriage virtually becomes an astigmatic dial, such as is recommended by Verhoeff (3), but adapted for use at the minimal reading distance.

This apparatus has been in use in the eye clinic of the Peking Union Medical College for more than a year. The results obtained from tests made with it are far more satisfactory than with any other method with which we are familiar. With proper precautions against faulty position of the apparatus when applying the test, and with a later adjustment of findings for an existing hypermetropia or myopia, one may accurately determine the maximal near point of accommodation, the range of accommodation, and the existence of

an astigmatism together with its axis, from a test of the accommodation applied prior to a cycloplegic examination.

REFERENCES.

1. HOWARD, H. J. "A new apparatus for testing accommodation." *Arch. of Ophth.*, 1920. Vol. 49, p. 182.
2. DUANE, A. "Studies in monocular and binocular accommodation with their clinical applications." *Amer. Jour. Ophth.*, 1922. V. 5, p. 865.
3. VERHOEFF, F. H. "The 'V' test for astigmatism, and astigmatic charts in general." *Amer. Jour. Ophth.*, 1923. V. 6, p. 908.

FUNDUS CHANGES IN HYPERTENSION WITH AND WITHOUT NEPHRITIS.

By Dr. AARON BARLOW, Philadelphia.

THE term hypertension with and without nephritis as used in this paper, refers to a condition characterized by persistent elevation of blood pressure of undetermined origin, with little or no reduction in kidney function as evidenced by elimination, concentration and blood retention tests, and in which, as a result of prolonged hypertension, the kidney becomes secondarily involved. This definition does not necessarily imply a negation of the theory that nephritis is the cause of hypertension in some cases, but merely excludes the kidney as an exciting factor in a group of cases designated as essential hypertension.

The etiology of essential hypertension has been a subject of lively discussion for many years. Focal infection, excessive meat eating, intestinal auto-intoxication, endocrine disturbances and the stress and strain of modern life have all been advanced as possible etiologic factors. When we try to get a clear picture of its exact pathogenesis, however, we find ourselves looking through nebulous media.

According to Kahler and Sallman (15), hypertension is caused by stimulation, central or peripheral, of the vasomotor nervous system. In the central group, the stimulation is in the vasomotor center. This stimulation may be of toxic, vascular (cerebral arteriosclerosis), or reflex origin. The ovarian dysfunction in the so-called climacteric hypertension is an example of the reflex form. In the peripheral group, the stimulation is in the vasomotor nerve endings of the peripheral vessels. Recently, Bordley and Baker (2) of Johns Hopkins advanced a new theory, based on the experiments of

Anrep and Starling (1) of London, and upon their own findings.

The London investigators have found, in their experiments on animals, that a relative anemia of the vasomotor center results in a compensatory rise in the systemic arterial pressure. The Baltimore pathologists have found arteriosclerosis constantly present in the medulla oblongata, in patients presenting a history of hypertension; in the presence of normal blood pressure they found the arterioles in this region unaltered. They therefore suggest that an arteriosclerotic process in the medulla oblongata may be the essential factor in the production of persistent high blood pressure.

On the other hand, we have the contention of many that the sclerotic changes in the vessels are secondary to, and apparently the result of hypertension. However, whether arteriosclerosis causes hypertension, or vice versa, it is generally accepted, I believe, that hypertension is accompanied by arteriosclerosis, in at least some portions of the body. Since the retinal vessels can be studied minutely and their progressive changes noted with the ophthalmoscope, the ophthalmologist, often called upon by the internist for aid in diagnosis and prognosis, is naturally keenly interested.

In this connection, the findings of O'Hare and Walker (24) are of interest. In a series of 100 cases, they studied the relationship of peripheral arteriosclerosis (radials, brachials and temporals), retinal arteriosclerosis and hypertension. They found cases with peripheral arteriosclerosis which had no retinal arteriosclerosis and no hypertension. On the other hand in cases where retinal arteriosclerosis and hypertension were present, they found no peripheral arteriosclerosis.

To quote these observers: "Our results definitely establish the fact that the peripheral vessels play little or no part in hypertension. They do show, however, a definite relationship between small vessel sclerosis indicated in the retinal arteries, and high blood pressure. . . . We must assume that this disorder of small vessels is more or less general. We cannot think for an instant that hypertension is associated only with sclerosis of the retinal arteries. These are probably, therefore, a fair index of small vessels throughout the body." They emphasize the great importance of examining

carefully the retinal vessels instead of merely glancing at the fundi for hemorrhages and white spots. Headache is a prominent symptom in hypertension. The laity has learned to associate this symptom with eye strain, headache frequently being the only complaint which brings the patient to the oculist. Since the fundus changes in hypertension are fairly characteristic, it is one of the diseases which is often first discovered by the ophthalmologist. [It is especially important to think of this condition when a patient in middle life, a time hypertension is most likely to occur, comes to us complaining of headache and dizziness. An examination which does not include a careful study of the retinal vessels is incomplete.]

While the changes that take place in the ocular fundus in hypertension and nephritis have been well described in text books and monographs, it is by no means always easy to decide from a fundus examination whether we are dealing with a case of hypertension with or without nephritis. Perhaps the best classification of the fundus changes in cardiovascular renal disease is that of Wagener (32) of the Mayo Clinic.

He divides them into four groups:

(1) Arteriosclerosis of the hypertension type and hemorrhages without actual retinitis.
(2) Arteriosclerotic retinitis.
(3) Retinitis of hypertension plus nephritis.
(4) Retinitis of nephritis.

He defines each of these as follows:

1. In a certain number of cases of hypertension there occurs in association with retinal arteriosclerosis a few scattered retinal hemorrhages without retinal edema and without exudates. These do not constitute a definite retinitis, could readily be produced by hypertension with local vessel changes, and do not necessarily need an added toxemia as a factor in their causation.

2. With more advanced local vessel-wall changes, there may occur, besides hemorrhages, small areas of exudation, or degeneration which appear ophthalmoscopically as small bright white spots usually grouped around terminal arteries, especially in the macular region. In places where the lumen

of the artery has been completely or practically obliterated in a portion of its course, proliferation of new vessels also takes place. Thus a picture is produced which is designated as arteriosclerotic retinitis, and which is probably the result of severe endarteritis, and by no means typical of, or necessarily associated with nephritis.

3. In another group of cases, in a retina which often shows only a mild or moderate degree of arteriosclerosis, there appears slight edema of the optic disc, scattered hemorrhages of flame-shaped or round deep type, and a few white patches of soft-edged exudate with slight surrounding edema, the so-called cotton-wool exudates.

For the production of these acute changes, a toxic factor other than that concerned in the slow production of arteriosclerosis would seem to be necessary. The kidney has long been considered the source of this toxemia, and hence this type of lesion has been designated the retinitis of hypertension plus nephritis.

4. Finally, there are seen fundi in which the outstanding feature is hemorrhages and marked edema of the retina, the latter resulting in measurable swelling of the disc, "snow bank" exudate in the retina around the disc with perhaps localized patches of increased density, cotton-wool exudates, and often a star figure in the macular region. These changes constitute the classical "albuminuric retinitis." They may occur without previous arteriosclerosis in the retinal vessels, and are hence designated the retinitis of nephritis.

It may be said then that the outstanding feature in the fundus picture of hypertension is the change in the retinal vessels. This change is characterized by a reduction and irregularity in the caliber of the arteries, indentation of veins by crossing arteries, narrowing and increased brilliance of the central light streak, loss of translucency of the arterial walls, tortuosity of the arteries and increased caliber of the veins. Of these changes, the first two mentioned, namely, contraction of the arteries and arterio-venous compression are the most important. According to O'Hare and Walker (24), these two signs are positive evidence of arteriosclerosis, while the other signs, they feel, are too variable and too dependent of personal judgment of what is normal and what is pathologic.

My own findings are in entire accord with this observation, and I would emphasize arterio-venous compression as a most valuable sign. Of course, when the compression is slight and indefinite, hypertension can only be suspected, but a deep indentation with the veins appearing as if cut in half by the crossing artery is, in my experience, an almost pathognomonic sign. A diagnosis of hypertension in the presence of this phenomenon will almost invariably be confirmed by the sphygmomanometer.

In addition to these vascular changes a few scattered retinal hemorrhages may be present, but they are by no means an essential feature in the fundus picture of hypertension. Should the hypertension persist and the retinal arteries become more and more sclerosed, the circulation and nutrition of the retinal elements will be proportionately disturbed, leading to hemorrhage and such tissue changes as exudates and secondary degenerations. Thus the fundus picture of the second group in this classification, namely, arteriosclerotic retinitis, is produced. What takes place in the retinal arteries must undoubtedly also take place in the arteries of other organs, the kidney not excepted. Fishberg (11), in a recent study of the character and distribution of the arterial changes in seventy-two cases of essential hypertension, found that the arterioles of the kidney were involved in 65%; the pancreas in 50%; the liver in 30%, and the brain in 20%. The change in the renal vessels leads to impairment of kidney function.

When, in addition to the retinal arteriosclerosis of hypertension, there appears, in the fundus, edema of the disc and retina, scattered hemorrhages of flame-shaped or round deep type, and a few patches of cotton-wool exudate, we have indications of renal disease causative of the retinal lesions. This is the fundus picture of retinitis of hypertension plus nephritis. It will thus be seen that the fundus pictures in arteriosclerotic retinitis, and in retinitis of hypertension plus nephritis have many features in common, such as arteriosclerosis, hemorrhages, white spots, etc., and are sometimes most difficult to differentiate. The chief differential feature, however, is the degree of arteriosclerosis.

In arteriosclerotic retinitis, the sclerosis is quite marked, the arteries sometimes appearing as fine white lines, empty of

blood; while in retinitis of hypertension with nephritis the vessel sclerosis is of a moderate degree. Benedict (6) draws a sharp distinction between the contraction of the retinal arteries in chronic hypertension and that in arteriosclerotic retinitis. In hypertension, the reduction in the caliber of the arteries is more uniform; the arteries are not beaded; and there is little or no tendency toward the formation of perivascular plaques or the proliferation of new vessels. Also, in arteriosclerotic retinitis, Moore (18) found the condition unilateral in 45% of his cases.

While this classification is a very admirable one, it must not be expected, of course, that every case will conform to it. Complicated pictures are often encountered which are very puzzling and confusing. Wagener and Keith (33) have reported fourteen cases of marked hypertension with neuroretinitis and adequate renal function. The swelling of the disc varied from 1–6 D. There were flame-shaped and deep retinal hemorrhages with cotton-wool exudates, and in seven of these cases the retinal picture was that of "albuminuric retinitis." I have recently seen a case of a somewhat similar nature. The patient was a woman of about 45. There was a typical star-shaped figure in the macula in both fundi with several small gray spots of exudate above the disc, and a few small superficial hemorrhages. There was no clinical nor laboratory evidence of nephritis. The urine at times showed a trace of albumin with an occasional hyaline cast; elimination was good as proved by the phenolsulphonthalein test, and the blood showed no urea nitrogen retention. The blood pressure, however, was 230 systolic and 110 diastolic. The retinal arteries were contracted and there was definite arteriovenous compression. The medical diagnosis was "essential hypertension." When one sees cases of this nature one is naturally confronted with the much discussed and as yet imperfectly understood subject of the pathogenesis of the fundus changes in nephritis. A paper of this sort would not be complete without a brief consideration of this very interesting subject.

From a genetic aspect, the fundus changes in the diseases under consideration may be viewed from the standpoint of (*a*) changes in the retinal vessels and (*b*) changes in the ret-

ina itself. As regards the changes observed in the retinal vessels, some authors maintain that they are due to a primary arteriosclerosis, while a majority of the more recent writers are of the opinion that a general vasoconstriction is responsible not only for the contraction of the retinal arteries, but also for the so-called compression phenomena, retinal hemorrhages, etc. Benedict believes that the thickening of the arterioles and capillaries which has been designated by Gull and Sutton as arteriocapillary fibrosis is secondary to a persistent hypertension.

With regard to the retinal lesions generally associated with nephritis, the discussion in recent years has centered chiefly upon the question whether the retinitis is secondary to the nephritis and therefore an indication of the extent of kidney damage, or whether certain other factors, toxic or circulatory, are mainly concerned in the production of both retinal and kidney lesions.

While the occasional occurrence of visual disturbance as a symptom in nephritis has been observed since Bright first described this disease exactly a century ago, the first ophthalmoscopic picture of albuminuric retinitis was described by Heymann in 1856, five years after the discovery of the ophthalmoscope by Helmholtz.

The literature on the subject has since grown to enormous proportions, and contains many divergent views and theories. Many of the more recent writers feel that this divergence of opinion is due, in no inconsiderable measure, to the large amount of disagreement exhibited in clinico-pathological literature in classifying the various types of nephritis and their relation to arterial disease.

It was formerly believed that the fundus lesions were produced by toxic matter resulting from disintegration of kidney tissue which, circulating in the blood, exerted an evil influence on the delicate, highly specialized and easily destructible retinal tissue, and on the retinal vessels. The introduction of newer methods in the study of kidney disease and kidney function, particularly blood chemistry, has caused many, especially French authors, to believe that the fundus lesions are produced by retained toxic substances in the blood, such as urea nitrogen, resulting from renal insufficiency. Thus,

according to Rochon-Duvigneaud (25), "The diseased kidney is the cause of the retinal lesion . . . in consequence of deficient elimination and the retention of urea or some substance intimately related to urea." However, retinal changes characteristic of nephritis, even the classical picture of albuminuric retinitis can occur without urea-nitrogen retention. Such cases have been reported by a number of observers. These, together with the cases reported by Wagener and Keith (33) and my own case previously mentioned in this paper, would tend to upset this theory.

Schieck and Volhard (29) do not favor the toxic theory. They even go so far as to assert that the retinal lesions are never directly due to nephritis. Volhard says: "If we ask ourselves to which remote effect of kidney disease the retinal lesions stand in compulsory relation, we must say, not to toxic uremia, azotemia and disturbance of kidney function, not to the remote effect of edema . . . not to the degree of albuminuria and presence of casts . . . not to the qualitative and quantitative change in the urinary secretion, but singly and alone to the most important and remote symptom of kidney disease, the origin of which is still entirely concealed in darkness, namely, to the rise in blood pressure."

Volhard believes there is a definite relationship between high blood pressure and the occurrence of retinitis, and that retinal changes occur only in the cases of nephritis in which there is hypertension. According to this author the retinal changes, instead of being secondary to those in the kidney, occur simultaneously with them. He believes that the general arteriolar constriction causes an ischemia of the kidney as well as of the retina, and that the retinal changes are the result of the angiospastic ischemia. This theory, according to Cheney (9), would explain the typical star-shaped figure sometimes observed in cases of choked disc with hypertension, whereas the toxic theory fails to offer any explanation.

While it is true that a majority of the patients with retinitis have or had hypertension, exceptions are not uncommon. Retinitis has been observed in cases with little or no hypertension. And again, as Moore (18) states, retinitis is often absent though the blood pressure is greatly raised. According to Kahler and Sallman (15), fundus changes in es-

sential hypertension and in nephritis occur only and exclusively in such cases in which the rise in blood pressure is due to a stimulation, toxic or vascular, of the vasomotor center (central hypertension), and never in association with peripheral hypertension. If the hypertension is vascular in origin, the vascular fundus changes predominate, and if toxic in origin, the retinal changes predominate. The same authors have also found an increase of cholesterin in the blood in many of their cases, and they therefore suggest that this may be an additional causative factor in the formation of the white spots in the retina.

Recently, Martin Cohen (7) has revived the old conception that the main cause of the retinitis is to be found in vascular changes. He maintains that a primary arteriosclerosis is the basic pathologic factor in the production of the retinal lesions in nephritis. In a series of eighteen cases, he studied the fundus ophthalmoscopically and then examined the eyes microscopically. He found instances which showed no arteriosclerosis ophthalmoscopically, but retinal arteriosclerosis was present in the same patients when examined miscroscopically.

Also choroidal arteriosclerosis was present without retinal arteriosclerosis. He found "nothing definitely pathologic except the arterial changes and causing as a result whitish spots, hemorrhages and papillitis."

That arteriosclerosis may be the immediate cause of albuminuric retinitis was first suggested by Duke Carl Theodore about forty years ago, and later by Michel, Weeks and others.

Schieck (27), however, in 1907 showed two cases of albuminuric retinitis in which there was no evidence of vascular changes. Retinitis occurs in pregnancy, occasionally in acute nephritis, and during the World War retinitis without any noticeable change in the vessels of the fundus was a frequent complication in many of the cases of so-called trench-nephritis. Cohen (7) evidently is impressed with the difficulty of explaining all the clinical fundus changes in all forms of kidney disease on the basis of arteriosclerosis, for he admits "it is possible that, in the course of glomerular nephritis, certain unknown factors produce arterial changes." He insists, however, that a vascular wall disease is the basic pathologic factor.

Others have taken a middle course and, like Slocum (31), believe "that while it seems to be more than probable that the etiology of the kidney disease is mainly the cause of the associated retinitis, nevertheless, a possible etiologic relation between the associated characteristic retinitis and the nitrogen retention and toxemia of advanced nephritis cannot be denied absolutely."

Finally, there are those who, like Batty Shaw (26), Behan (3) and others refuse to accept hypertension, urea nitrogen retention or vascular sclerosis as the sole cause of the pathologic changes in the fundus. Moore (18) summarizes Batty Shaw's (26) theory as follows: "The changes in the retina are dependent neither upon a disease which is primarily in the kidney nor upon changes in the retinal vessels, but upon a toxin in the blood, the nature of which has not as yet been determined, which attacks both kidney and retina alike. As evidence that changes in the retina can be produced by a disordered blood state rather than by changes in the kidney or in the retinal vessels, he mentions eclampsia, where changes may be seen in the eye and in no other organ, and which are remedied by termination of pregnancy. He believes that arteriosclerosis is a first effect, and arteriosclerotic retinitis a later one, of a toxemia which acts slowly and in minimal quantity, and that when the toxemia acts quickly or in accumulated large bulk the changes of renal retinitis are produced."

Behan (3) maintains that sclerosis of the retinal vessels represents the evidence of nature's defense rather than a causative factor in the production of retinitis. He believes that some injurious substance in the blood stream is the basic cause of any or all of the signs noted in the retinitis of nephritis. He concludes as follows: "In the cause of the kidney pathology, we have the cause of the eye change. The ocular pathology does not exist because of the kidney change any more than the nephritis is the result of the eye change."

BIBLIOGRAPHY.

1. Anrep and Starling. "Central and reflex regulation of the circulation." *Proc. Roy. Soc. London*, 1925.
2. Bordley and Baker. "A consideration of arteriosclerosis of the cerebral vessels and the pathogenesis of hypertension." *Bull. Johns Hopkins Hosp.* 38:320 (April), 1926.

3. BEHAN, J. L. "Fundus changes in nephritis." *Trans. Sect. on Ophth.*, 1922.
4. BENEDICT, W. L. "Retinitis of cardiovascular and of renal diseases." *Am. J. of Ophth.*, 4:495 (July), 1921.
5. BENEDICT, W. L. "Retinitis of Acute Nephritis." *M. Clin. of N. Am.*, 5:275 (Sept.), 1921.
6. BENEDICT, W. L. "Retinitis of hypertension plus nephritis." *Trans. Sect. on Ophth.* 1922.
7. COHEN, MARTIN. "Significance of pathologic changes in the fundus." *Trans. Sect. on Ophth.* 1922.
8. COPPS, L. A. "Retinal arteriosclerosis." *Am. J. of Ophth.* 1921. V. 4, p. 810.
9. CHENEY, R. C. "Toxemia of pregnancy from an ophthalmoscopic standpoint." *Trans. Sect. on Ophth.* 1924.
10. DAWSON, LORD. STARLING, E. H., et al. "Discussion on Hyperpiesia." *Brit. M. Jour.*, London, 1925. II, 1161–1170.
11. FISHBERG, A. M. "Anatomic findings in essential hypertension." *Arch. Int. Med.* 35: 650 (May) 1925.
12. GILBERT, W. "Ueber Augenerkrankungen bei Nieren und Gefässleiden." *Münch. Med. Woch.* 1921., V. 68, p. 979.
13. HANSSEN, R. UND KNACK, A. V. "Zur genese der Retinitis Nephritica." *Klin. Monatsb. f. Augenh.* 1917. V. 59.
14. HORNIKER, E. "Augenspiegelstudien bei Kriegsnephritis." *Graefe's Arch. f. Ophth.* 1921. V. 105, p. 104.
15. KAHLER, H. AND SALLMAN, L. "Ueber die Netzhaut. Veränderungen bei Nieren und Gefässerkrankungen (Zugleich ein Beitrag zur Pathogenese der Retinitis Nephritica)." *Ztschr. f. Augenh.* Berlin, 1925. LVII, 386–423.
16. MAJOR, R. H. "Relationship between certain products of metabolism and arterial hypertension. *J. Am. M. Ass.* 1924. LXXXIII, 81–84.
17. MAJOR, R. H. "The possible relationship between guanidine and high blood pressure." *Am. J. M. Soc.* 170: 228 (Aug.), 1925.
18. MOORE, R. F. *Medical ophthalmology.* 1925.
19. MOSCHCOWITZ, E. "Hypertension: Its significance; relation to arteriosclerosis and nephritis and etiology." *Am. J. Med. Soc.* (Nov.) 1919. p. 668.
20. MOSCHCOWITZ, E. "Clinical and anatomic relations in chronic nephritis." *Arch. Int. Med.* 26: 259 (Sept.), 1920.
21. MOSCHCOWITZ, E. "Pathology of hypertension." *J. Am. M. Ass.* 79 (Oct. 7), 1922.
22. MOSCHCOWITZ, E. "Problems in renal pathology." *Am. J. Med. Soc.* (Oct.), 1926.
23. VON MONAKOW, P. "Blutdrucksteigerung und Niere." *Deutsches Arch. f. Klin. Med.* 133: 129 (Aug.), 1920.
24. O'HARE, J. P. AND WALKER, W. G. "Arteriosclerosis and hypertension." *Arch. Int. Med.* V. 33 (March), 1924.

25. ROCHON-DUVIGNEAUD. "Albuminuric retinitis." Abstracted in *Ophth. Rev.* 1912. XXXI, 307–310.
26. SHAW, H. B. Discussion on "The significance of the vascular and other changes in the retina in arteriosclerosis and renal disease." *Proc. Roy. Soc. Med.*, 1922–1923. V. 16, Part 3.
27. SCHIECK, F. "Ueber retinitis albuminurica." *Ophth. Ges.* Heidelberg, 1907.
28. SCHIECK, F. "Die genese der retinitis albuminurica." *Klin. Monatsb. f. Augenh.* Vol. 66. 1921.
29. SCHIECK, F. UND VOLHARD, F. "Netzhautveränderungen und Nierenleiden, Zentralbl. f. d. " *Ges. Ophthal. u. Grenzgeb.* 5. 465: 481. 1921.
30. SLOCUM, GEO. "A study of ophthalmoscopic changes in nephritis." *J. A. M. A.* 67, (July) 1916, p. 5.
31. SLOCUM, GEO. Abstract of Discussion on Behan, Jos. L. "The fundus changes in nephritis." *Trans. Sect. on Ophth.* 1922.
32. WAGENER, H. P. "Retinitis and renal function in cardiovascular renal disease." *Am. J. of Ophth.* V. 7, p. 292. (April) 1924.
33. WAGENER, H. P. AND KEITH, N. M. "Cases of marked hypertension. Adequate renal function and neuro-retinitis." *Arch. of Int. Med.* V. 34. 1924.
34. ZUR NEDDEN. "Experimentelle Untersuchungen ueber spezifische Beziehungen zwischen Netzhaut und Nieren." *Bericht der Ophth. Ges.* Heidelberg, 1908.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A clinical meeting of the Section took place at St. Bartholomew's Hospital on Friday, March 11th, the President, Mr. Ernest Clarke, occupying the chair.

Cases.

Exophthalmic Goiter with External Ocular Paresis.

Mr. Foster Moore showed a woman with this condition. Five years ago he saw her with severe exophthalmic goiter, for which part of her thyroid was removed. At that date she had marked proptosis, and most of her ocular muscles were paralyzed, though not the internal branches. There was still marked ocular palsy at this date, and when reading she had to move her head from side to side in order to follow the print. Her general health, however, had become very much better. He regarded the condition as muscular.

Chronic Ophthalmoplegia and Ptosis.

Miss Rosa Ford showed a girl suffering in this way. She was aged 17, and was otherwise quite healthy. The first sign was noticed seven years ago, at which date when she looked at an object she closed one eye with her hand, probably because of diplopia, which was still present. So little inconvenience was felt that advice was not sought until four years

later, the occasion being the refusal of her on an application to enter the Civil Service. Now, there was partial ptosis, and almost complete ophthalmoplegia in both eyes. The right eye was the more prominent, the left being a little divergent. In the right eye the vision was $\frac{6}{6}$, in the left, $\frac{6}{9}$ partly. The fundi looked normal, except for some pallor at the outer halves, and there was slight cupping—probably physiological—of the outer half of the right disc. Pupil reaction to accommodation on both sides was sluggish. She was able to do her work, that of typing, without difficulty. Some 47 cases of the kind had now been collected from the literature, but no cause for the condition had yet been assigned. Messrs. McMullen and Hine, in their paper on the subject, seemed to incline to the view that it owned a nuclear origin, believing that there existed a congenital weakness of nerve centres leading to premature decay.

Mr. Lindsay Rea spoke of a case of his own in which the movements were limited only when the patient turned her eyes to the left; when looking in front of her, the muscle balance appeared to be normal. That seemed to point to a nuclear, not a muscular, origin. His patient said the deficient movement of that eye occurred before there was any sign of exophthalmic goitre coming on.

Bilateral Kerato-irido-cyclitis.

Mr. Davenport showed this as what he hoped was the end-result of a very severe inflammation of the anterior part of the eye. The patient was a female child, whose appearance made one suspicious of tuberculosis, but, though the signs at her apex seemed to confirm this idea, she was not considered at Brompton Hospital to be certainly tuberculous. When first she was seen she had intense inflammation of the lower half of the cornea, there was much k.p. and the irides were studded with nodules. After a residence in the country the condition largely subsided, the k.p. had disappeared, and there remained an interstitial nodule in each cornea, and dotted spots in each iris.

Mr. Davenport also showed a case of degenerative changes in the cornea, in a glycosuric patient.

Buphthalmos in a Boy.

MR. SCOTT showed a boy with buphthalmos. His mother said the left eye had always been large, and that the left pupil seemed larger than the right. When the boy was first seen, three weeks ago, the tension in that eye was raised, and there was pulsation of the retinal arteries to be seen, on and near the disc. Now, the tension was not raised, and no pulsation of arteries was apparent.

MR. M. S. MAYOU said this was an unusual case, as it was rare for buphthalmos to be unilateral. When it occurred unilaterally it was usually associated with neurofibromatosis on the same side. This boy's face was not quite normal, for it was somewhat thickened over the jaw, and the fold of the top lid was obliterated. He believed there was some proptosis on the affected side. The buphthalmos in this case, therefore, he regarded as a mild form of neurofibromatosis.

Macular Choroido-retinitis.

MR. SCOTT showed a case of left macular choroido-retinitis in a woman. The patient said the sight of her affected eye was normal, until she was 19 years of age, and now she had a large pigmented scar in the macular region, while in the forward part of the vitreous was what he took to be an infiltration of the canal of Clouquet. It was of inflammatory nature, a so-called macular coloboma.

Facial Cleft, Hare-lip, and Ectropion.

MR. ACKROYD showed a patient who presented this combination of defects, and various suggestions for operative procedures were discussed.

The Pectin in an Owl.

THE PRESIDENT brought an owl, the pet of his daughter, in order to demonstrate to members the pectin, which took the place of retinal vessels in the bird, and supplied nourishment to the retina. He understood that the theory that the

pectin was used for accommodative purposes was no longer largely held. It was believed by Miss Mann and others who had written on the subject that the pectin gave to the bird an acuter vision than it would have if it possessed retinal vessels, as the pectin came off from the optic nerve and did not obstruct the line of vision.

Other cases were shown.

Report of Section on Neurology.

At a meeting of this Section held on Thursday, February 10th, the subject of "Optical Nystagmus" was discussed. The chair was occupied by the President, Sir James Purves Stewart, K.C.M.G.

Dr. E. Arnold Carmichael read the opening paper. He said that most of the literature on the subject was in a foreign language. There were several different types met with in pathological states, such as congenital, spontaneous, miners', vestibulo-central, and occasionally cerebral nystagmus. There were also groupings according to the site of the lesions, *i.e.*, high spinal lesions, medullary lesions, cerebellar lesions, vestibular lesions, mid-brain lesions, lesions of retina, and in certain congenital anomalies, such as albinism. Dr. Carmichael said he would deal with the nystagmus, which could only be evoked by special methods in normal individuals, and which in certain pathological states could not be elicited. On a railway journey a nystagmus was set up by watching the passing landscape. In nystagmus there were two phases; a slow movement of the eyeballs in one direction, a quick movement in the opposite direction. The direction of a particular nystagmus movement was named according to its quick component. It was essential, the author said, to make records of the amplitude, the rate, the duration of the nystagmus; also to note whether, in the quick phase, the eyeballs returned to the central axis, or beyond it. A valuable paper on the subject had recently been published by Fox & Holmes. There were records of patients with a large central scotoma and but little peripheral vision left still having the power of manifesting optic nystagmus; others with one eye blind and only small peripheral vision in the other, who yet could have optic

nystagmus on both sides. This suggested that a lesion or lesions of the optic nerve or chiasma in no way interfered with production of nystagmus if some peripheral vision remained. Barany reported two cases of hemianopia in which there was no nystagmus to the blind side, and in which the suggestion therefore was that the lesion was in the region of the optic radiations. A lesion affecting the optic radiation and causing hemianopia might result in loss of nystagmus to the hemianopic side. Lesions in the higher visual centers were frequently associated with loss of nystagmus to the opposite side. Lesions of the frontal lobe, especially in the motor area of the eye, caused loss of nystagmus to the opposite side.

It was possible to map out the pathways probably involved in the production of optic nystagmus. It was not necessary to have an intact optic tract or optic nerve; neither was an intact optic radiation essential; therefore it was fair to assume that with only one visuo-sensory path it was possible to obtain optic nystagmus to both sides. Lesions above one occipital lobe caused loss of nystagmus to the opposite side. Therefore it was probable that from the region of the occipital cortex, an association bundle of fibers passed, these being situated in front of the occipital pole; and that the occipital cortex on one side was connected with the higher visual centers of the opposite cerebral hemisphere. An intact pre-Rolandic center was necessary for the production of nystagmus. Lesions of the optic radiations might or might not cause a loss of the reflex. Lesions on the sensory path caused no loss of nystagmus, but lesions affecting the motor path did so.

No theory of the mode of production of optic nystagmus had yet been brought forward which would account for all the conditions. Nystagmus had been produced in a baby 8 hours old, therefore neither myelination nor fixation could be looked upon as conditions necessary for bringing it about. A stimulation by irritation of the vestibular nerve on both sides at one time caused a complete disappearance of the nystagmus.

Conclusions.

Dr. Carmichael briefly stated his conclusions as follows: (1) Nystagmus was lost to the opposite side in lesions which

were situated in the higher visual cortex, in the frontal lobe, in the association bundles connecting the higher visual centers and frontal lobes. (2) Nystagmus might or might not be absent when lesions of optic radiations were present. (3) Optic nystagmus was unaffected by lesions of the optic tracts, optic chiasma and optic nerve, provided the patient had some sight remaining. Hence (4) nystagmus depended on a pathway situated in the cerebral hemisphere. (5) Nystagmus was affected by disorders of the vestibular apparatus. (6) Slight vestibular dyspunction might be recognized by changes in the optic nystagmus.

Discussion.

The President said it must be taken that nystagmus was a phenomenon which was present in normal persons; and that the more abnormal they became from lesions in various parts of the visual apparatus or in the cerebral hemispheres, the less likely were they to exhibit the ordinary optic nystagmus. Evidently the mechanism of normal optic nystagmus was as follows. When looking out of a moving railway train a person caught sight of an object in the periphery of his visual field and at once made a voluntary effort to bring it into the center of the field. Without the hint of an object in the periphery, there was no stimulus towards the effort which resulted in the nystagmus. He asked Dr. Carmichael whether it was possible that a lesion of the intermediate fibers in the centrum ovale, leading to the frontal lobe might, in the future, have an analogous diagnostic significance to lesions of the visual fibers in the temporal lobe. It was important to exclude cases of oculo-motor palsy, because if a patient was unable to look in a certain direction, he was unable to show nystagmus on the side.

Sir James Dundas-Grant said he understood that when vestibular nystagmus was present, the induction of optic nystagmus would neutralize it, in other words, optic nystagmus had a stronger effect than the vestibular. Some writers considered that the center for optic nystagmus was Deiter's nucleus. He showed a cold-air apparatus which he was in the habit of using in his aural work, and suggested that it

would be less irksome than employing cold water when carrying out such investigations as Dr. Carmichael had done.

Dr. Cloake said he did not quite see the force of the arguments with which Dr. Carmichael dismissed Barany's view of the phenomena of nystagmus. Dr. Carmichael said nystagmus was elicitable in patients who had only very limited peripheral vision, also in patients who had large central scotomata, therefore it could not be due to the voluntary attempt to follow the object across the field of vision, and quick restoration so as to seize upon the oncoming stripe. Once the normal cerebral mechanisms were in force, he, the speaker, thought the obliteration of parts of the field of vision could not be expected to alter greatly the attempt made to follow an object once it had been caught sight of. Barany's view of the phenomenon was a common-sense view, and it was not easily dismissed.

Dr. Carmichael, in reply, said optic nystagmus was not always lost even though a patient was hemianopic. He only proceeded to test vestibular nystagmus because some of the people he was testing for the purposes of this paper complained of giddiness. Barany's view was that the quick companion of the nystagmus was due to the oncoming stripe in the test and the desire to turn to look at it. But if the patient had but a small bit of peripheral vision left, the oncoming stripe could not be seen, therefore Barany was not right in that.

Discussion on the Value of Recent Methods of Treatment in the Late Stages of Ocular Syphilis.

A joint meeting of the Sections on Ophthalmology and Neurology took place on February 11th, when the subject named above was discussed. The chair was occupied by the President of the Section on Ophthalmology, Mr. Ernest Clarke.

Mr. J. Herbert Fisher opened the debate. He first passed in review the different methods of giving or using the essential drug, mercury, and the supplementary use of iodide of potassium for the later manifestations. But practitioners at a later date seemed content to administer gray powder in tabloid form. Unless these pellets were crushed before being

swallowed, they were often passed as taken, unaltered. And iodide of potassium was given in too many cases in only 3 to 5 gr. doses when 30 grs. was nearer the requirement. He thought such a discussion as this should help a closer working association between ophthalmic specialist, physician, neurologist and general surgeon. In some cases of enlarged glands, arthritis and bone disorders which were regarded as tuberculous could have been seen to be syphilitic if the eyes had been examined. Ehrlich's discovery of salvarsan and neo-salvarsan seemed to have eliminated the dangers of the optic atrophy which had followed the use of the earlier atoxyl and soamin. The trouble was that salvarsan or neo-salvarsan were too often regarded as sufficient in themselves, and mercury was erroneously left out of account. The Wassermann reaction was not infallible, it was indeed but a supplement to the well-established methods of clinical observation. The difficulty of obtaining the original arsenical preparations led to many attempts being made to manufacture substitutes, some being apparently even innocent of arsenic. With the establishment of numerous V. D. clinics through the country, which were largely resorted to, the disease was better understood and observed, and the public had gained a better idea of its ravages. Cases of ophthalmia neonatorum were becoming more rare in hospital practice.

In the late manifestations of syphilis in the eye he would include iritis (sometimes the nodular variety), scleritis, disseminated choroiditis, interstitial keratitis; these apart from the disease in which the ophthalmologist joined forces with the neurologist, ophthalmoplegia. He would also include diffuse neuro-retinitis with dust-like opacities in the vitreous. This was now less frequent, he thought, than 30 years ago, perhaps because of more thorough treatment of the primary stage. In secondary syphilitic iritis, the new injection treatment had been of the greatest assistance in preventing damage to the eye, probably because the antidote was brought into direct contact with the vascular iris. Interstitial keratitis from acquired syphilis he had not seen earlier than five years after the primary infection; it might not occur until fifteen or twenty years after. As contrasted with the case of inherited syphilis, he had not seen the second eye attacked when the

disease was acquired. In interstitial keratitis from inherited syphilis there was no evidence that treatment by either the older or the newer method arrested or even modified the progress of the disorder.

It would be interesting to hear from the neurologists concerning the present frequency of tabes and of primary optic atrophy in locomotor ataxy. In regard to the last-named, he had been impressed with the way in which well-conducted mercurial treatment would delay the advance of the atrophy of the optic nerve fibres, especially if a fair visual field had been preserved. He had seen the Argyll-Robertson pupil phenomenon disappear under mercurial treatment. Aneurysms might also be expected to be less frequent now, also sclerotic changes due to the disease which resulted in nuclear palsies and other focal lesions. Gummatous meningitis had always been amenable to mercurial administration.

Summarising, Mr. Fisher said the modern methods enabled more rapid control to be obtained of the primary and secondary manifestations of the disease: in regard to the eye, they were particularly useful when the disease affected the vascular tissues of the eyeball. It was hoped that the remote effects of syphilis (tabes and G. P. I.) would be less disastrous now that the newer methods were being so largely used, and especially as patients were coming earlier and more thoroughly under treatment.

Dr. Purdon Martin opened from the neurological side. He said there were three main types which late syphilis of the nervous system assumed, though in any patient these might be combined in varying degree. Syphilis might affect principally the parenchymatous elements in the main mass of the brain or spinal cord, and the common ocular symptoms were the Argyll-Robertson phenomenon, and probable complete paralysis and irregularities of the pupil in the absence of other oculomotor symptoms. Syphilis might affect principally the adventitious elements in the brain, causing what was known as cerebral syphilis, an accompaniment of which was, often, papillœdema. The associated ocular palsies were not commonly of central causation. Or the disease might affect primarily the elements lying outside the main mass of the central system. He went on to refer to the external ocular

palsies and primary optic atrophy. He had seen one case of optic atrophy in which it was likely the new preparations had done harm, and it seemed likely that tryparsamide might precipitate optic atrophy. He considered that chemical treatment was of little avail in acute cases of optic atrophy. A positive Wassermann confirmed a diagnosis, but a negative Wassermann in the blood of a patient of 50 or over meant little.

SIR ARNOLD LAWSON concentrated his remarks on optic atrophy, a condition which he had found to be almost hopeless from the treatment point of view. All one could hope to do was to arrest the progress of the atrophy. Organic arsenic compounds he regarded as harmful, while for mercury in this condition he had nothing but contempt. The only treatment which seemed to hold out any hope seemed to be large doses of iodides, but even this form of treatment was profoundly unsatisfactory. He believed the reason was that in primary optic atrophy one was dealing with a primary degeneration as opposed to a secondary degeneration following on the effects of active inflammation. It did not seem reasonable to expect a cure of optic atrophy once the process had started; all one could hope for was an arrest of it. He had found that at least 25% of cases of late syphilis did not give a positive Wassermann reaction, though it was well known that the symptoms suffered from were due to syphilis. Of 109 men admitted to St. Dunstan's, 62 had a positive Wassermann, 20 had a negative one, while of the remainder 27 were so bad that treatment was impossible. Most of the men had undergone intensive treatment at other hospitals before coming to St. Dunstan's, but it seemed to have made them no better than those who had not had such treatment. Many of the patients died; some had mental conditions, G. P. I., meningitis. He had come definitely to the conclusion that one could not hope to arrest this optic atrophy by chemicals, and that the sooner that idea was relinquished, the better would be the results attained. In this disease sufficient recognition had not been made of the great mainstay of all treatment, skilful, scientifically applied rest. It was valuable in all forms of impaired function. The hope, in his view, lay in securing these cases of primary optic atrophy early and giving them

sound sanatorium treatment, on the lines adopted for tuberculosis.

Mr. S. H. Browning spoke of having treated large numbers of cases of interstitial keratitis by the new methods, *i.e.*, salvarsan, neo-salvarsan and bismuth. He mostly, however, combined this treatment with mercury. These cases did better with intensive anti-syphilitic treatment than without it. For tabes there should be a blending of the old treatment with the new. He had a good word for the Aachen treatment in the case of those able to bear the expense. He had had success in the use of bismuth instead of mercury.

Col. L. W. Harrison held a somewhat hopeful view of the treatment of interstitial keratitis due to congenital syphilis, a view which was supported by medical officers at treatment centers. At Rochester Row Military hospital there were decent results obtained from sub-conjunctival injections of salvarsanized serum. He dealt with the treatment of syphilis in a general way, and spoke of the experiences at the St. Thomas' Hospital V. D. clinic.

Dr. Pines (of Leningrad) spoke of good results from sodium iodide intravenously in late syphilis, especially in interstitial keratitis. None of the patients so treated showed iodism.

Dr. Gordon Holmes referred specially to the optic atrophy associated with post-syphilitic diseases (tabes and G. P. I.). He did not think optic atrophy invariably led to blindness; a considerable number of cases of optic atrophy of tabetic origin preserved their vision for years, especially under treatment. It had been shown that the essential lesion in tabetic optic atrophy in G. P. I. was an interstitial syphilitic process associated with a primary degeneration of a certain number of nerve fibers. In a number of examinations he had confirmed this: there was a considerable interstitial change; a cellular infiltration in the nerve, generally passing in from the pia, usually along the small vessels, and so into the nerve. The lesion was nearly always found in the region of the optic thalamus, or at least it was most intense there. From observation of series of cases treated respectively by mercury and arsenical preparations, he had concluded that the arsenical preparations were relatively ineffective. In order to secure results, treatment by mercury should be carried out.

MR. LINDSAY REA gave accounts, with pictures of fundi, of a number of cases he had treated by the newer methods, and described the results.

DR. A. FEILING dealt at some length with the subject from the neurological standpoint.

MR. FISHER, in reply, said he felt convinced that thorough mercurial treatment delayed the progress of primary optic atrophy in tabetics. As a result of more thorough and earlier treatment, he thought the cases of congenital syphilis now seen were milder than formerly. He agreed with Sir Arnold Lawson's advocacy of measures to promote the patient's best possible state of general health.

REPORT OF THE SECTION ON OPHTHALMOLOGY, NEW YORK ACADEMY OF MEDICINE.

By Dr. THOMAS HAYES CURTIN, Secretary.

MEETING OF FEBRUARY 21, 1927. DR. CLYDE E. McDANNALD, CHAIRMAN.

Dr. Franklin Bracken (by invitation) presented a case of **Tower skull with optic atrophy.** This case presents some interesting features of oxycephaly in that the vision is not reduced nor the fields contracted in proportion to what one might expect from the appearance of the nerve heads and the deformity of the orbits. He has a marked exophthalmos and is able to converge very little and with difficulty. The X-ray of the skull taken by Dr. H. A. Goalwin shows very shallow orbits. The inclination of the outer walls to the median plane of the head (as observed in the top view) is 68°, the average normal being 45°. The depth of the orbit is 1 *cm* less than the average orbit. The right optic foramen is somewhat pear-shaped and slightly larger than average, measuring 3.9 x 5.1. The left is smaller, measuring 3.0 x 3.9 but there is not sufficient change in the bony canal to cause much pressure on the nerve. The left nerve is decidedly pale, presenting a neuritic atrophy; but the right is so masked by medullated fibers that one can see very little of it. The fields are practically normal; and vision with correction is R $\frac{20}{20}-$, L $\frac{20}{20}-$.

Discussion: Dr. Schoenberg says that the most interesting lesson in these cases is an early diagnosis. Schloffer has devised an operation consisting of a decompression of the optic canal, and has reported a few cases with appreciable improvement. He also states that the time to operate is when an active process is present, with papillitis or optic neuritis, before the stage when the optic atrophy sets in.

DR. THOMASSON: I have a case now under observation, who when first seen had optic atrophy. However, strange as it may seem, vision and mentality have improved.

DR. BERNARD SAMUELS gave a lantern slide demonstration of **implantation cysts of the orbit, sclera, cornea,** anterior and post chambers, as well as of the iris. In addition he presented slides of cysts of the pigment epithelium of the iris and of cystic degeneration of the retina. The last preparation shown was of an enormous cystoid cicatrix at the limbus following a trephine.

DR. A. H. THOMASSON read the first paper of the evening, entitled, **The development of the glaucoma scotoma.** (Published in full, pages 319–27 of this number.)

DISCUSSION: DR. KNAPP: Dr. Thomasson's painstaking exposition of the visual field changes in glaucoma illustrates their importance in the consideration of a glaucomatous process and how essential they are in the proper management of these cases. In fact, in addition to the tonometer there has been no contribution which has added so much to our knowledge of glaucoma as scotometry; and I think Col. Elliot's emphasis on this examination is as important a contribution as the trephining operation. The development of the arcuate scotoma is interesting. I would like to ask Dr. Thomasson whether he has any explanation for this beyond the presence of the vessels at the disc margin and why is central vision finally involved by the nasal extension of the scotoma?

DR. SCHOENBERG: I would like to ask Dr. Thomasson if he can explain the development of the scotomata, whether they develop along the vessels or by pressure on the optic nerves. Also, have these various types different prognoses?

DR. THOMASSON: I have developed few theories. For the most part I have followed Elliot. Answering Dr. Knapp, why scotoma developed in the nasal direction—We do not know. Dr. Elliot, however, believes that these fibers are the longest and are affected first. Dr. Schoenberg asks why sometimes vertical scotoma developes and sometimes the scotoma is in the horizontal meridian. Answer.—We do not know. Fibers below the disc are the heaviest and more numerous. The prognosis is bad in all cases. Types that develop macular degeneration soonest, naturally have the worst prognosis, so

the horizontal type is the worst type, Elliot calling it central scotoma of glaucoma.

DR. ARNOLD C. KLEBS, Nyon, Switzerland (by invitation), read the second paper of the evening, entitled, **Historical perspectives in ophthalmology.** (To be published in full.)

MEETING OF MARCH 21, 1927. DR. CLYDE E. MCDANNALD, CHAIRMAN.

DR. WILLIAM BROWN DOHERTY. **Presentation of an anterior chamber irrigator.** This consists of a globular upper extremity with a small flattened bluish tip. It is designed to maintain a steady and even flow of solution and to prevent the introduction into the eye of any foreign material or air bubbles. The tip is colored a dark blue so that the entrance into the anterior chamber is easily seen. The instrument is very light and is not cumbersome.

A second model is being made with a ball valve glass attachment on the side near the angle of the tip which will allow the instrument to be rapidly refilled.

DR. BEN WITT KEY. **Case of post-operative cyst of the iris.** I brought this same case before you at the January meeting of this section, when the iris cyst was observed, and I asked your advice as to the management of it. For the benefit of those who did not see the case at that time, this drawing of it may serve as an illustration.

It was my understanding that it was most important to remove every portion of the cyst as well as the iris pillars from which it seems to spring. Of course the method of procedure in removing it was the important feature of the case clinically. Whether or not to make a keratome incision boldly into the cyst, making a protecting conjunctival flap with the same incision, or whether with a Graefe knife to make an incision in the peripheral limbus or in the plane of the roots of the iris, were the questions in mind at the time.

Realizing the delicacy of the cyst structure and the necessity for protection against vitreous loss, since it appeared to be present in the anterior chamber, I attempted the removal of it in the following manner.

1st. A small but fairly long triangular conjunctival flap

was made—thus—and a suture placed, a fairly good bite of the conjunctival margins being caught up by the suture.

2d. At the roots of the iris and passing across the base of the cyst for two-thirds of the extent of the cystic area an incision was made with a small Graefe knife, at the same time securing a narrow conjunctival flap.

3d. This incision was completed with Stevens scissors extending well over the roots of the opposite pillar.

4th. The right iris pillar was then incised well back into its free portion, and then released,—when the left iris pillar was treated in the same manner. The right iris pillar was then grasped in iris forceps and dissected free with cyst substance from beneath the conjunctival flap and without event. At the moment when the cyst portion was being removed, still attached to both iris pillars, it was felt that the cyst wall was attached to the posterior surface of the cornea and the traction produced a slight dimpling of the cornea at this point, but with iris scissors and Ziegler knife this was dissected free and the cystic structure was removed still attached to both pillars of the iris.

5th. The conjunctival suture was now tied, bringing the lips of the wound firmly in position. With fine dressing forceps the pigment and other débris were removed and the new iris pillars stroked in place with the spatula.

No complications have arisen and I have brought him here for your inspection. His vision with correction—$\frac{10}{10}+1$. It is of course too soon to assume that every portion of the cyst was removed, but it seems reasonable to believe that it was, until time shall prove it to the contrary.

Dr. Julius Wolff presented (I) cases of **Plastic operation for restoration of eyebrow.** E. K., 17 years old. In early childhood was burnt in region of right brow, resulting in a scar with complete destruction of the hairs of the brow, except a few at the outer end. Left eyebrow intact and heavy. To replace the missing right brow a free graft was taken from the left brow, consisting of a strip of skin 1¾ in. long, ¼ inch wide and of the full thickness of the cutis. It contained the hairs of the lower half of the left eyebrow, and the upper and lower incisions converged in the depth, so that the flap was triangular in cross section. This made it fit accurately into a single

incision made for its reception in the region of the right brow. To give the hairs the right direction the flap was turned 180 degrees from its original position. Accurate coaptation was made of the edges of the flap and the skin by closely placed, superficial fine sutures. A compressive dressing was left in place five days and the graft made a complete "take."

(II) **Perforating injury resulting in cyst of iris.** H. G., 35 years old. In 1916 operation for removal of traumatic cyst in the left eye. A few years later presented a shrunken capsular membrane adherent to iris and increased tension. Iridectomy performed. When seen in 1924 the cyst had recurred and filled $\frac{3}{4}$ of the anterior chamber. It was not removable, so a filtering operation was performed at the lower outer corneal margin at the seat of the cyst, so as to drain the same subconjunctivally. By this operation the tension has been kept down, pain has been relieved and the eyeball thus far preserved, but a large subconjunctival cyst has formed under the lower lid about as large as a small bean. The cyst in the anterior chamber is collapsed, as it is draining through the trephine wound.

DISCUSSION: DR. AYRES reported a similar case in which he operated on the eye subconjunctivally by the Elliot trephine. This is just a makeshift to take the place of enucleation. The man still retains his eye.

DR. JOHN VIRDEN presented a case of **Bilateral keratoconus.** Patient whose vision four years ago with glasses was $\frac{10}{10}$, now reads $\frac{10}{100}$.

DR. BERNARD SAMUELS gave a **Lantern slide demonstration of Microscopic slides of sarcomas and gliomas.** The tumors were of various sizes and shapes, pigmented and non-pigmented. In several of them the tendency to necrosis was beautifully brought out. Especially interesting were the circumpapillary sarcomata.

Of glioma both the endophytum and exophytum types were presented. In one eye two primary tumors were shown. Dr. Samuels dwelt on the fact that while extensive necrosis is characteristic of glioma yet the adjoining tissues do not show the violent reaction so frequently observed in necrosis occurring in sarcoma.

The second paper of the evening was **A discussion of some of**

the more common ophthalmic operations, by DR. F. H. VERHOEFF, of Boston (by invitation).

After making some remarks upon the subject of ophthalmic operations in general, the speaker described some of the procedures that had impressed him as especially valuable during his operative experience of twenty-seven years. In connection with strabismus operations, he pointed out that there should always be taken into consideration the facts that tenotomy causes exophthalmos and limitation of motion while advancement of a muscle causes enophthalmos and astigmatism. He now believes that a partial tenotomy is never indicated—if a tenotomy is required at all it should be complete and its effect limited by a suture if necessary. The advancement operation he still prefers is the one he devised when a medical student. The so-called resection operations and tucking operations are inferior to it in important respects.

The operative treatment of acute and chronic dacryocystitis was discussed. In acute cases he employs the Agnew incision. In chronic cases with fistula, he packs the sac with cotton and then extirpates.

Enucleation of the eye he always performs under local anesthesia except in cases of young children. The needle is inserted into the orbit through the upper lid in three places. He always implants a glass ball except in cases of old people. Only one suture is employed, by means of which Tenon's capsule is overlapped in four layers in front of the ball. The ocular tendons are not included in the suture. An artificial eye is inserted and a pressure bandage applied.

In cases of contracted socket, the cavity is distended by means of a glass ball under a pressure bandage and later shaped by means of an artificial reform eye also held in place by a pressure bandage. Skin grafting should be employed as a last resort.

For ptosis, he prefers Reese's operation when the levator is entirely inactive. Otherwise, he prefers resection of half the tarsus combined with advancement of the levator.

In cases of trichiasis resulting from trachoma, extirpation of the tarsus usually relieves the condition. In especially bad cases, scalping of the lid margin is the best procedure.

For exposure keratitis with infection, following Gasserian

ganglion operations, he has found a conjunctival flap remarkably effective. It is allowed to remain indefinitely, or until the lacrymal secretion returns, when it is easily removed with a spitula, leaving a clear cornea.

For acute glaucoma he does a Reese iridectomy unless the anterior chamber is too shallow in which case he makes the incision with a narrow Graefe knife and leaves a conjunctival bridge. For chronic glaucoma he employs a modified iridotasis. A button-hole is made in the iris midway between the pupil and the limbus, the iris grasped at the upper margin of the hole and pulled out beneath the conjunctival flap. In this way the sphincter is allowed to remain within the eye, so that the pupil is displaced slightly if at all. This is also the best operation for buphthalmos.

The speaker then described his method of performing iridectomy in operations for cataract. In conclusion he briefly described a new method he had recently devised for removing cataracts in their capsules, and for suturing wounds of cataract operations.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M.D., F. A. C. S., New Rochelle, N. Y.

(*Continued from March issue.*)

VIII.—THE SENSE OF SIGHT AND REFRACTION.

128a. Abramowicz, A. **Contraction of the visual field in pregnancy.** *British Journal of Ophthalmology*, Jan., 1927.

129. Cavara. **Clinical studies concerning inverse astigmatism.** *Bollettino d'oculistica*, July, 1922.

130. Lo Cascio. **The astigmatism of aberration in the human eye.** *Annali di Ottalmologie e Clinica oculistica*, February, 1923.

130a. Chase, A. E. **Color blindness in industry.** *J. A. M. A.*, xxxvii., 1058, 1926.

130b. Dykman, A. B. **Changes in refraction in diabetes mellitus.** *Amer. Jour. of Ophth.*, ix., 825, 1926.

130c. Ellett, E. C. **Refraction changes.** *J. A. M. A.*, lxxxvii., 1453, 1926.

131. Elschnig. **Changes of refraction in diabetes mellitus.** *Medizint Klinik*, January, 1923, p. 17.

131a. Graves, Basil. **Change of tension in the lens capsules during accommodation and under the influence of various drugs.** *Brit. Med. Journal*, Jan., 1927.

132. v. Hippel, E. **Myopia.** *Festrere zur Jahresfeier der Universitae. Goettingen*, 1923.

133. Holm, Eyler. **Central and eccentric fixation.** *Acta ophthalmologica*, 1923, p. 49.

134. Jackson, Edward. **Lasting paralysis of accommodation after using cycloplegics.** *American Journal of Ophthalmology*, March, 1925, p. 207.

134a. Lancaster, Walter B. **The fusion faculty and some of its anomalies.** *Amer. Jour. of Ophth.*, ix., 247, 1926.

135. LASSIGNARDIE and MANINE. **Shrapnel bullet lodged in the left occipital lobe near the angular convolution productive of right homonymous lateral hemianopsia and word blindness.** *Annales d'oculistique*, clx., 9, p. 719.

136. LAWSON, SIR ARNOLD. **Tints and their value.** *British Journal of Ophthalmology*, November, 1924.

137. LEVINSOHN. **The question of the genesis of myopia.** *Klin. Monatsbl. f. Augenheilkunde*, lxxi., p. 205.

137a. LEUDDE, W. H. **Mechanism of accommodation.** *Amer. Jour. Ophth.*, x., 15, 1927.

138. LUTZ. **Uniocular hemianopsia of central origin.** *Annales d'oculistique*, clx., 4, p. 265.

138a. O'BRIEN, C. S. **Retinoscopy with cylinders.** *Amer. Jour. of Ophth.*, ix., p. 107, 1926.

139. MAJEWSKI. **A case of spastic pseudomyopia.** *Revue générale d'Ophtalmologie*, October, 1923.

139a. POST, LAWRENCE. **Changes in refraction in diabetes mellitus.** *Amer. Jour. of Ophth.*, ix., 666, 1926.

140. v. SAPPERN, H. **Achromatopsia in homonymous hemianopsia with full vision.** *Klin. Monatsbl. f. Augenheilkunde*, lxxi., p. 101.

141. SHOJI. **Photochemical study of the absorption of ultra-violet rays while passing through the media of the eye.** *Annales d'oculistique*, clx., 5, p. 356.

142. DE VINCENTIIS. **The blind spot in the normal and the diseased eye.** *Annali di ottalmologie e clinica oculistica*, October-December, 1922.

143. WITTE, O. **Myopia.** *Zeitschrift f. Augenheilkunde*, li., p. 163.

Out of a total number of 28,094 cases of refractive error CAVARA (129, **Clinical studies concerning inverse astigmatism**) found that 5,241 had inverse astigmatism. According to his figures inverse astigmatism is more common than is ordinarily thought, and is present in a quarter of all astigmatic eyes. The form in which it is most frequently met with is the compound myopic, the form in which it is the least common is the mixed. Simple and compound inverse myopic astigmatism is more common than inverse hypermetropic astigmatism. With regular astigmatism the reverse is true. The frequency of inverse astigmatism increases with age, it being found in 7.62% of all astigmatic eyes in patients under 10 years of age, and in 85.7% of those over 80. The frequency of regular astigmatism decreases with age. After discussing the various theories concerning the cause of this phenomenon the writer ascribes the predominating rôle to the action of the lateral recti together with the diminishing

elasticity of the cornea. Inverse astigmatism is usually of slight degree, not over 1 D. It exceeds 3 D in only 2%. As age advances not only the frequency, but the degree increases. The axis coincides with the horizontal meridian in 59.98% of the cases; less often it forms an angle to the meridian, the percentage growing smaller as the angle becomes larger. The deviation of the axis downward and outward is somewhat more common than upward and outward; the reason for this is unknown. As age advances the number of eyes with horizontal axes increases. Inverse astigmatism is rather more common in the right eye than in the left; the reverse is true of regular astigmatism. In fifty per cent. of all patients with inverse astigmatism the error of refraction is confined to one eye; when both eyes are affected the axes are symmetrical in two-thirds of the cases, and the degree is the same in two-thirds of the cases. The statistics show no difference between the sexes. Finally Cavara emphasizes the points that inverse astigmatism need not always be congenital, that it may be developed during life, and that modifications in degree and in the inclination of the axes are possible in the course of years.

Lo Cascio (130, **The astigmatism of aberration in the human eye**) says that in ametropic eyes a certain degree of astigmatism is produced which cannot be detected by retinoscopy, but only through a subjective examination. For this reason subjective tests are always needed to supplement and correct the retinoscopic findings.

Ellett (130c, **Refraction changes**) describes the different conditions that may bring about a change in the refraction of an eye. Each cause is supplemented by case histories from his practice. A cycloplegic was used to estimate the error of refraction, except those cases where accommodation was no longer a factor or where the drug was not necessary.

Yudkin.

Dykman (130b, **Changes in refraction in diabetes mellitus**) finds that the most plausible explanation for the manifestation is a change in the lenticular tissue due to an alteration in the physical and chemical composition of the eye fluids. This hypothesis is in accord with that of other observers particularly Duke-Elder. When the patient is ill with dia-

betes the refractive error is of a myopic tendency and under treatment, changes to hypermetropia and finally reverts to the original error of refraction as the bodily fluids return to the normal state. YUDKIN.

ELSCHNIG (131, **Changes of refraction in diabetes mellitus**) reports a case of diabetes in which the hypermetropia increased about 2 D before dietic treatment was instituted. The urine contained 7% sugar and acetone. In another case he showed that the change of refraction must take place in the lens, as it did not take place in one eye, which was aphakic, while it did occur in the other. Finally he quoted a third case as evidence that fairly sudden increase of hypermetropia may take place in old age without the presence of diabetes.

POST (139a, **Changes in refraction in diabetes mellitus**) reports two cases that revert to the former refractive error when placed on a diabetic diet. He explains this process on Duke-Elder's hypothesis of the osmotic changes in the lens caused by a variation of the molecular concentration of the blood and tissue fluids due to the varying sugar content. YUDKIN.

HIPPEL (132, **Myopia**) is inclined to think that near work does not cause myopia and that the so-called school myopia is in fact hereditary. He also thinks that in consequence of the various degrees of curvature of the cornea it is by no means necessary that a myopia should always be associated with an elongation of the axis of the eye.

In opposition to Hippel's views LEVINSOHN (137, **The question of the genesis of myopia**) pointed out that there are difficulties which cannot be overcome in the way of the theory that myopia is a purely hereditary anomaly. The influence of near work has, he thinks, been proved beyond question.

WITTE (143, **Myopia**) presents some statistics from the Greifswald clinic. Total number of patients, 34,753. Myopes, 13.8%; 16% men, 11% women, but myopias of 10 D and upwards were more common in women. The lower degrees of myopia were met with most frequently among persons accustomed to near work; the higher degrees among those who were not so accustomed. The number of complications increase with the degree of myopia and were pres-

ent in 4.2% of the men, and 7.7% of the women in all cases. Divergent strabismus was the most common. The higher the degree the worse the vision, taking into account only the uncomplicated cases. The vision was more reduced in those who did not do near work and in women than in near workers. The results agree perfectly with the statistics formerly obtained by Siebenlist under like conditions from the same clinic.

MAJEWSKI (139, **A case of spastic pseudomyopia**) reports a case of this kind which had been under his observation for several years. The patient had worn a −7 sph. before each eye since he was 16 years old. Under atropine the retinoscope showed a slight hypermetropic astigmatism to be present. The spasm yielded only as long as the effect of the atropine lasted, even when the administration of this drug was continued for several weeks. It was not until the onset of presbyopia that the patient was freed from the spasm, so that he could see well in the distance without glasses.

HOLM (133, **Central and eccentric fixation**) says that in fixation it is not a certain point, but an object the image of which falls upon the region of the fovea, that is fixed. Patients with pathological changes confined to the center of the fovea fix large objects well. With small objects they employ a somewhat lateral fixation. Also in adaptation to the dark, when central fixation is impossible because of a central scotoma, there is paracentral or parafoveal fixation.

JACKSON (134, **Lasting paralysis of accommodation after using cycloplegics**) reviewing the literature finds no justification for attributing lasting paralysis of accommodation to the use of cycloplegics particularly in refraction. The few cases cited have no detailed information so that it is impossible to say whether the condition is caused by cycloplegic or is just a coincidence. ALLING.

LANCASTER (134a, **The fusion faculty and some of its anomalies**) believes that in carnivora and some of the arboreal group there is a certain amount of binocular fusion and that the power of suppression is probably absent because they possess the faculty of great motility of the head to scrutinize the field of vision. In man, where the macula is well developed and the field of vision is approximately 180°, the ability of

suppression is necessary to escape an intolerable situation caused by muscular imbalance or refractive errors. The power of fusion may be lacking in some individuals and corrective exercise or glasses will not remedy the ocular disturbance, but where it is held in abeyance or is slightly imperfect, it is possible to develop this faculty by treatment.

YUDKIN.

CHASE (130a, **Color blindness in industry**) finds that the Eldridge-Green type of lantern is the only apparatus with sufficiently broad and accurate application which can be used by industrial concerns to detect the greatest percentage of colored blind satisfactorily. The usual tests by worsteds alone eliminates only 50% of the color blind. The tests should be made by a competent worker, preferably an ophthalmologist.

YUDKIN.

A great deal of confusion and lack of accurate appreciation concerning the value of tinted glasses is current at the present time. The normal human eye is well protected by the cornea and lens with regard to the ultra-violet rays, no rays of shorter wave length than 350*mm* being allowed to pass these structures. The eye is less well protected against the luminous and infra-red rays, the pigment of the iris and choroid and the contracting power of the pupil being the only protection.

It must be conceded that human eyes are adapted to endure without injury the ordinary daylight of the "home" country. There are certain accessory and complicating factors. These are (1) the intensity of the light, (2) the duration of the exposure, (3) the presence of glasses, (4) occupation or trade.

With regard to the relative value of tints experiments were carried out and one fact immediately became apparent, namely that, irrespective of the tint, the thickness of the glass employed was often of the greatest importance. Two thicknesses of glass were used, one of 0.5*mm* and the other of 2*mm*, the latter causing much greater absorption of light. LAWSON (136, **Tints and their value**) suggests that in advising tinted glasses, the thickness should be specified as well as the tint.

The following tints were used in the experiments:—

Crooks—A, A2, B, B2.

Amber—1, 2, 3.
Fieuzal, 1, 2, 3, 4, 5.
Peacock Blue.
London Smoke—2, 4.

The experiments showed that Peacock Blue gave by far the greatest protection for both the red and infra-red rays, and good protection for the violet. Fieuzal gave the greatest protection against the violet end of the spectrum. Crooks protected well against the violet end and, in the deeper shades, (B and B2) gave fair protection against the red end of the spectrum. The advantage of the Crooks lens was the uniformity and regularity of the increasing protection from the lighter to the deeper shade. Fieuzal was most irregular in this way, probably owing to lack of uniformity in manufacture. London Smoke gave good protection at the red end. Crooks B gave good protection throughout the whole range of the spectrum.

With regard to wearing tinted glasses, as stated before, Lawson emphasised the fact that a normal eye in its normal surroundings should need no protection. In extraordinary circumstances, such as mountaineering or in the glare of the tropics, it is generally the luminous and heat radiations against which protection is needed. Lawson would recommend Fieuzal tints, or, in extreme cases, Peacock Blue. In physical disease and in neurasthenia the tolerance to light may be reduced, and for such cases Crooks A or A1 may be used. This glass has the advantage of not grossly interfering with the general color scheme, but wearing such a protection should not be encouraged.

In cataract due to glass blowing it has been shown that the infra-red rays are to blame, and in these cases Peacock Blue should afford the best protection. Lawson recommends Crooks A or A2 in cases of incipient cataract, and thinks this important. After extraction protection should be given, as the eye is now less well protected against the violet and ultra-violet rays, and Lawson advises Crooks A or London Smoke or Fieuzal. In active inflammatory disease of retina or choroid Peacock Blue should be ordered. P. G. DOYNE.

LASSIGNARDIE and MANINE (135, **Shrapnel bullet lodged in the left occipital lobe near the angular convolution productive**

of right homonymous lateral hemianopsia and word blindness) give an account of a soldier wounded during the war. The bullet was removed from the brain nearly a year after the wound was received. Recovery from the operation was uneventful. The preceding examination of the eyes revealed normal conditions except for the hemianopsia with intact macula. The vision of each eye was 2–3. While the patient could write fairly well, he had much difficulty in reading. Manuscripts he could decipher with difficulty word by word. Single letters he recognized easily, but he could make out words only by the help of the letters. He read his own name fairly easily through attention to the first letters. Pictures and objects he recognized quickly and easily, giving them their correct names. He solved easily simple problems in arithmetic. Writing from dictation was rapid, but copying from print was slow with large letters, while copying a manuscript was less difficult. He could read and write the notes of music. No change in this condition followed the operation.

The trouble in both of the cases reported by SAPPERN (140, **Achromatopsia in homonymous hemianopsia with full vision**) was apoplexy with right sided hemianopsia. At first there was total blindness, but this passed off except for the color blindness. This still persisted in one case up to the time of the last examination, while in the other color perception suddenly returned at the end of ten days with normal color fields. Even large colored objects were taken to be green in both cases.

LUTZ (138, **Uniocular hemianopsia of central origin**) says that a hemianopsia may exist in one eye while the visual field of the other is perfectly normal. The cause may be intraocular, it may be due to glaucoma, detachment of the retina, or occlusion of large retinal vessels, or it may be produced by a lesion in any part of the optic tract from the lamina cribrosa to the calcarine fissure. A uniocular hemianopsia by itself is of no use in the localization of a lesion. This is very rarely to be found in the intraorbital portion of the optic nerve, or in Gratiolet's optic radiation; in the majority of cases it occurs near the chiasm, especially in the part of the nerve between the optic foramen and the chiasm. The hemian-

opsia is almost always due to an extracerebral cause. Its uniocular nature is explained by the fact that the decussating and non-decussating fibers form separate bundles and end in different cell layers of the corpora geniculata externa and the cortical visual centers. It may be permanent or transient, and the final condition of a binocular or monocular amaurosis may be a bitemporal or homonymous hemianopsia. Or the reverse may take place and a hemianopsia may pass over into an amaurosis.

By the application of the mathematical formulas for the determination of the powers and axis created when two cylinders are placed at oblique axis.

O'BRIEN (138a, **Retinoscopy with cylinders**) shows why the retinoscopic shadows are produced in some of the following errors of refraction. If the sphero-cylindrical error in the eye is corrected with a combination of cylinder and a sphere, the point of reversal of motion with the retinoscope is obtained. If the sphere is correct but cylinder under-corrected and at the proper axis, one meridian is at the point of reversal, but the original band of light continues as before with the exception that the sphere is more brilliant and more rapid in its movement. If the sphere is correct but cylinder over-corrected and at the proper axis, one meridian is at the point of reversal but the original band of light is reversed. If the sphere is correct and the cylinder correct as to focus, but off axis, in hyperopic astigmatism, a with-band and an against-band is present, the with-band being at approximately 45° to the opposite side of the proper axis and an against-band at right angles to this; in myopic astigmatism, the against-band in this case acts in the same manner as the with-band in hyperopic astigmatism. If the sphere is correct but the cylinder under-corrected and off axis, a with-band of light which lies to the opposite side of the proper axis and within a few degrees of it and, as usual the against-band at right angle. In myopic astigmatism the same development of an against and a with-band with the against-band lying less than 45° to the opposite side of the proper axis, is noted. If the sphere is correct but the cylinder over-corrected and off axis, there is a with-band of light which lies to the opposite side of the proper axis but more than 45° away from it. In myopic astigmatism the against- and with-

bands appear; the against-band lying far from the proper axis and the with-band as usual, at right angles.

YUDKIN.

SHOJI (141, **Photochemical study of the absorption of ultra-violet rays while passing through the media of the eye**) says that the absorptive power of the media of the eye to ultra-violet rays is dependent on their albumin content. The lens has the greatest power of absorption, then follow the cornea, vitreous, and aqueous.

DE VINCENTIIS (142, **The blind spot in the normal and the diseased eye**) says that in the normal eye the blind spot is usually an oval with a vertical axis, never a horizontal, and very rarely is a circle. The oval form is due partly to the anatomical form of the papilla, partly to an astigmatism. The horizontal breadth of the blind spot in the normal eye varies from 4° to 6° 30′, the vertical from 6° to 8° 30′. An enlargement of the diameter more than a degree is to be considered suspicious. The distance from the fixation point to the middle of the blind spot is about 15°. The absolute scotoma is surrounded by a zone amblyopic for color about 1° broad; when its breadth is over 2° it is to be considered pathological. The first color to be perceived, starting from the blind spot, is blue, then almost simultaneously, red and green. The size of the blind spot is not materially changed by myopia or hypermetropia of less than 8 D, but in high myopia it appears to be smaller, in high hypermetropia larger, also in myopic conus of high degree, a papillary diameter or more, which may be projected in its exact form. For the origin of Bjerrum's scotoma in glaucoma the writer thinks the rôle of the vessels important in so far as the nerve fibers which accompany the large vessels at the scleral ring are the first to suffer from the simultaneous intraocular and endovasal pressure. In choked disc the blind spot is enlarged uniformly in all directions, so it retains its form; the enlargement increases proportionally with the papillary change, and is caused by the alteration of the peripapillary nerve fibers. In simple neuritis the blind spot itself is not changed, but the neighboring amblyopic zone is almost constantly enlarged, most markedly in a vertical direction. In sympathetic inflammation a relative color scotoma may be observed about the

blind spot, which disappears after the subsidence of the inflammation. Finally, in commencing tabetic atrophy there is an enlargement of the peripapillary amblyopic zone.

These observations by GRAVES (131a, **Change of tension in the lens capsule during accommodation and under the influence of various drugs**) were made upon a rather remarkable eye. This eye had been injured by an intraocular foreign body. A small hole had been made in the anterior Lens capsule, and the foreign body could be seen lying at the bottom of the anterior chamber. The lens matter had been completely absorbed and there was left the anterior and posterior lens capsules, the intervening space between the two capsules being filled with aqueous fluid. The media were absolutely clear and vision was $\frac{6}{6}$ with a + 14 DS lens. There was no sign of any inflammatory reaction or thickening of the lens capsule. With such an eye it was possible to examine the condition of the lens capsule with the slit-lamp under varying conditions of accommodation, and also under the influence of drops, such as atropine and eserine.

The ordinary state of the capsules was as follows; they were slightly lax, the anterior slightly convex forward, the posterior a little wrinkled and mobile. When the eye looks into the distance, the capsules straighten and become taut. When the eye looks at a near object, the capsules become lax, the posterior much more so than the anterior. On touching the lids the capsules become lax.

The influence of a midriatic was to cause the capsules to become tense; the effect of a miotic was to cause the capsules to become lax.

Novocaine, though dilating the pupil, produced no effect on the tension of the capsules, neither did adrenalin.

The effect of eserine upon the eye, after it had been well atropised, was interesting. In this case the capsules began to become lax in an hour after the eserine instillation, but there was no marked lessening of the dilatation of the pupil 24 hours later.

Another point which Graves found was that the tension of the capsules caused by a midriatic is independent of the dilatation of the pupil also caused by the midriatic.

DOYNE.

Four general schemes are proposed by LEUDDE (137a, **Mechanism of accommodation**) for the study of the mechanism of accommodation; namely (1) the change in the anterior posterior diameter of the eyeball or a modification of the convexity of the cornea; (2) an increase in the convexity of the lens due to an inherent tendency to assume a spherical form when the tension is released from without,—this is the Helmholtz hypothesis and which the writer labels as the "intrinsic theory"; (3) an alteration of the form and position of the lens by external force—in contradistinction to the former this may be designated as the "extrinsic theory"; (4) a combination of the intrinsic and extrinsic theories. The first idea may be discarded for it has long been disproved by research. From the experimental work already done, the lens is definitely altered in the mechanism of accommodation. The curvature of the anterior part is more affected than the posterior. Applying the data from the literature and his observations on some experimental work he does not believe that a relaxation of the fibers of the Zonula of Zinn by the contraction of the ciliary muscle can produce the mechanism of accommodation. YUDKIN.

ABRAMOWICZ (128a, **Contraction of the visual field in pregnancy**) examined the visual fields in 81 patients in the last month of pregnancy. In two cases only did he find temporal contraction of the visual fields, and in one of these the temporal contraction was in the field of the right eye only. In three other cases he found temporal contraction to colored test objects only.

The alterations mentioned above occurred twice in primiparæ, once in second pregnancy and once in third pregnancy. The visual field returned to normal in all cases after delivery.

Of the 81 cases observed, 46 were primiparæ. Examination by X-rays showed no changes in the sella turcica in any of the cases.

He found no relation between the appearance and extent of the field contractions and the strength of the labor pains.

DOYNE.

IX.—THE CONJUNCTIVA AND CORNEA.

144. ALAND, A. H. **Ætiology and pathology of phlyctenular keratoconjunctivitis.** *American Journal of Ophthalmology*, December, 1925, p. 948.

144a. ALVIS, B. Y., and WEINER, M. **Treatment of trachoma by intramuscular injections of mercury.** *Amer. Jour. Ophth.*, x., 33, 1927.

144b. BEDELL, ARTHUR J. **Band keratitis.** *Amer. Jour. Ophth.*, ix., 887, 1926.

145. BLANCHARD. **Chancre of the conjunctiva of the upper lid.** *Annales d'oculistique*, November, 1923, p. 862.

146. CHARLIN. **Anaphylaxis to sulphate of copper in a case of trachoma.** *Ibid.*, clx., August, 1923, p. 630.

147. DENTI. **Chancre in the upper conjunctival fornix.** *Bolletino d'oculistica*, November, 1922.

148. DORSEY, J. G., and GILLETT, W. G. **Sarcoma of the caruncle.** *American Journal of Ophthalmology*, December, 1925, p. 942.

148a. DUNPHY, EDWIN BLAKESLEE. **Leptotrichosis of the conjunctiva.** *Amer. Jour. of Ophth.*, ix., 669, 1926.

149. FORT, A. C. **Observations on cause and treatment of vernal conjunctivitis.** *Journal of the Medical Association of Georgia*, xiii., p. 511.

150. FUNAISHI, SHINICHI. **Treatment of phlyctenular inflammation of the eye.** *American Journal of Ophthalmology*, August, 1925, p. 618.

150a. GARRETSON, W. T., and COSGROVE, K. W. **Ulceration of the cornea due to bacillus pyocyaneus.** *J. A. M. A.*, lxxxviii., 700, 1927.

151. GIFFORD, SANFORD R. **Marginal dystrophy of cornea.** *A. J. O.*, January, 1925, p. 16.

151a. GIFFORD, SANFORD R. **Epithelial dystrophy of the cornea and its relation to endothelial dystrophy.** *Amer. Jour. of Ophth.*, ix., p. 81, 1926.

152. GIFFORD, SANFORD R. **Eosinophilia and ætiology of Parinaud's conjunctivitis.** *Ibid.*, June, 1925, p. 450.

153. DE GONVEA. **A rare case of bilateral pannus of idiopathic origin.** *Annales d'oculistique*, clx., November, 1923, p. 882.

153a. GUY, W. H. **Interstitial keratitis in congenital syphilis.** *J. A. M. A.*, lxxxvii., 1551, 1926.

153b. HERSCHENDÖRFER, A. **Pigment changes in the conjunctiva of the lids in trachoma.** *Amer. Jour. of Ophth.*, ix., 812, 1926.

154. HOWARD, HARVEY J. **Rôle of the epithelial cell in conjunctival and corneal infections.** *American Journal of Ophthalmology*, December, 1924, p. 909.

155. KEY, BEN WITT. **Congenital zonular opacity of the cornea.** *Ibid.*, February, 1925.

155a. KEY, J. A. **Brittle bones and blue sclera.** *Archives of Surgery*, xiii., 523, 1926.

155b. KEY, BEN WITT. **Influence of protein therapy on experimental staphylococci infection of the rabbit's cornea.** *Amer. Jour. of Ophth.*, ix., 351, 1926.

156. KOYANAGI. **A new treatment for staphyloma. Injection of a concentrated salt solution into the vitreous.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., p. 499.

157. KRAUPA-RUNK. **Herpes corneæ.** *Zetischr. f. Augenheilkunde*, l., p. 345.

158. KREIKER. **Observation with the slit-lamp on the development of vascularization in the cornea.** *Hungarian Ophthalmological Society*, Budapest, February 4, 1923.

159. LANE, FRANCIS. **Extensive sarcoma originating from a pigmented nævus of the conjunctiva.** *American Journal of Ophthalmology*, September, 1925, p. 698.

160. LEHRFELD, LOUIS. **Allergic reactions in vernal conjunctivitis.** *Ibid.*, May, 1925, p. 368.

161. MORELLI. **A case of perithelioma of the bulbar conjunctiva.** *Bolletino d'oculistica*, December, 1922.

162. MORSE, SAMUEL. **Roseola of the conjunctiva.** *Journal of the A. M. A.*, April 25, 1925, p. 1256.

163. NORRIE. **Prophylaxis against blennorrhea neonatorum in Denmark.** *British Journal of Ophthalmology*, vii., No. 8.

163a. PATON, LESLIE. **The trigeminal and its ocular lesions.** *British Journal of Ophthalmology*, June, 1926.

164. PERRYRA. **Pseudotuberculosis of the bulbar conjunctiva.** *Bolletino d'oculistica*, July, 1923.

165. PILLAT. **Metastatic keratitis after epidemic cerebrospinal meningitis.** *Ophthalm. Gesellschaft*, Vienna, July 10, 1922.

166. SALLMANN. **Radium treatment of trachoma.** *Ibid.*, November 20, 1922.

167. SCHWARTZ, A. Q. **Nutritional keratomalacia.** *Journal of the A. M. A.*, lxxxv., p. 2025.

168. SHAHAN, W. E. **Neuropathic conjunctivitis.** *American Journal of Ophthalmology*, June, 1925, p. 461.

168a. SPAETH, EDMUND D. **Rotated island graft operation for pterygium.** *Amer. Jour. of Ophth.*, ix., 649, 1926.

169. VERHOEFF, F. H. **The cause of keratitis after Gasserian ganglion operations.** *A.J.O.*, April, 1925, p. 293.

170. WHITHAM, LLOYD B. **Epithelial dystrophy of cornea and ocular manifestations in diabetes.** *Ibid.*, October, 1924, p. 758.

171. WOLZ. **Hereditary nature of keratoconus.** *Archiv. f. Augenheilkunde*, xcii., p. 156.

172. YOUNG, C. A. **Electrothermophore in treatment of corneal ulcers.** *American Journal of Ophthalmology*, April, 1925, p. 293.

172a. ZIEGLER, S. LEWIS. **The surgery of trachoma.** *J. A. M. A.*, lxxxvi., p. 399, 1926.

CHARLIN (146, **Anaphylaxis to sulphate of copper in a case of trachoma**) reports the case of a 21-year-old patient who, after a month's treatment with the copper stick, developed a conjunctival hyperæmia, with photophobia and lacrimation. The duration of this trouble increased from a quarter of an hour to an entire day while the treatment was maintained, and at the same time with the local symptoms there was lowering

of the blood pressure and leucopenia. To render possible the continuance of the treatment for trachoma a desensibilization was brought about within seven months by the instillation of solutions of cupric sulphate, beginning at 1:10000 and increasing steadily in strength up to 1:10. Then it was found that the copper stick could be used again without the previous difficulty.

HERSCHENDÖRFER (153b, **Pigment changes in the conjunctiva of the lids in trachoma**) found pigment deposits in the scar tissue of the conjunctiva of four cases of trachoma. A histologic examination showed the pigment clumps under the epithelium in the cellular tissue, and in the tarsus, but no deposits in the epithelial cells. When the pigment appeared in a large mass, it was located extracellularly also. He believed the trachoma stains were produced by a process of oxydasis which was present in the diseased tissue. YUDKIN.

DORSEY and GILLETT (148, **Sarcoma of the caruncle**) report a case which was verified by histologic findings. Sarcoma of the caruncle is extremely rare. It is similar in its evolution to malignancy of this type elsewhere in the body.

ALLING.

LANE (159, **Extensive sarcoma originating from a pigmented nævus of the conjunctiva**) reports a case of recurrent epibulbar sarcoma invading both lids, originating from a pigmented nævus of the conjunctiva which was removed three years previously for microscopic study. He concludes that pigmented nævi of the conjunctiva are congenital growths which sometimes show a high potential tendency to become malignant. ALLING.

MORELLI (161, **A case of perithelioma of the bulbar conjunctiva**) found in a woman 24 years old a fleshy tumor about as large as a pea joined to the bulbar conjunctiva at the inner canthus by a short, slender pedicle. When the lids were closed it projected between them and interfered with the outflow of the lacrimal fluid. The histological examination of the growth proved it to be a perithelioma coming from the vessels of the conjunctiva. It was divided by connective tissue septa into seven lobules, was very vascular, and showed hyaline degeneration in its center.

FORT (149, **Cause and treatment of vernal conjunctivitis**)

ascribes vernal catarrh to hypersensitiveness to certain proteins or irritants. Following the plan of the American Hay-Fever Association he made diagnostic cutaneous tests with pollen and noted that all his fifteen cases gave positive reaction. Of those treated he had two cures and thirteen improvements. ALLING.

LEHRFELD (160, **Allergic reactions in vernal conjunctivitis**) studied the allergic skin reaction of five cases of vernal conjunctivitis. He regards the condition as an ophthalmic urticaria, or an allergic or atopic conjunctivitis because of the local and sometimes general eosinophilia, of its vasomotor disturbance, of its itching, and its allergic reactions.

ALLING.

GIFFORD (152, **Eosinophilia and ætiology of Parinaud's conjunctivitis**) divides the reported cases into four groups, those due to (1) Bacillus tuberculosis; (2) some form of leptothrix; (3) a virulent gram negative bacillus called microbacillus polymorphous necroticans by Pascheff, and similar in most properties to bacillus pseudotuberculosis rodentium found by Bayer and Von Herrenschwand; and (4) having negative bacteriologic finding. Gifford reports a case showing marked eosinophilia and belonging to the fourth group. ALLING.

PERRYRA (164, **Pseudotuberculosis of the bulbar conjunctiva**) observed in a boy 5 years old, one week after a pustular eruption on his right arm, a number of yellow nodules near the upper limbus which had developed with symptoms of a severe local inflammation. The bacteriological examination of an excised portion of the conjunctiva revealed a pseudotuberculosis bacillus belonging to group II of Wrede's classification. Histologically the nodules consisted of polynuclear leucocytes, giant cells, epithelioid cells and central caseous detritus.

DUNPHY (148a, **Leptotrichosis of the conjunctiva**) reports two cases in which the lesions were examined histologically and the organism was found. The disease was probably transmitted by close contact with a cat that had sore ears.

YUDKIN.

MORSE (162, **Roseola of the conjunctiva**) reports three cases of a subacute exanthematous conjunctivitis peculiar to secondary syphilis. The conjunctivitis of the exanthematous type is characterized by its suddenness of onset, lack of discharge, some photophobia and little or no lacrimation.

ALLING.

According to NORRIE (163, **Prophylaxis against blennorrhea neonatorum in Denmark**) the midwives in Denmark are required to instill a drop of a 1:150 silver nitrate solution in each eye of the child immediately after birth. The eyes are not washed out previously, but only wiped off with dry cotton or gauze. The effect of this measure is shown by statistics. During the years 1900–1904, when the prophylactic instillation by the midwives was not sufficiently insisted upon, sixteen children were blinded by ophthalmia neonatorum, while from 1915–1919 only one child suffered in this way in a population of 3,250,000. It must be remembered that the prophylactic measure does not prevent a late infection after birth.

According to SALLMANN (166, **Radium treatment of trachoma**) this treatment is of use only in the mildest cases. In severe cases the copper stick treatment cannot be replaced by radium. The combination ot the copper stick with radium is most valuable in the onset of recurrences.

SHAHAN (168, **Neuropathic conjunctivitis**) reports a case in which the diseased tissue was recognized by staining with mercurochrome after fluorescein failed to disclose the lesion, which at first seemed to be in the cornea. The stained area of conjunctiva was excised and a permanent cure was procured.

ALLING.

SPAETH (168a, **Rotated island graft operation for pterygium**) describes an operation in which the direction of the fibers of the pterygium is changed from the original horizontal to a perpendicular course by four incisions placed in the form of a rectangle and the tissue rotated 90°. The incised rectanglar piece of tissue is fixed with sutures placed in the four corners and supplemented by more sutures when necessary. He finds that the blood vessels and fibers atrophy and are replaced by cicatricial tissue by this island graft of conjunctival tissue.

YUDKIN.

ALAND (144, **Etiology and pathology of phlyctenular kerato-**

conjunctivitis) concludes that phlyctenular keratoconjunctivitis is definitely a disease of childhood and adolescence and one of the most frequent of all eye diseases in dispensary and free clinics. The pathological and bacteriologic studies indicate that it has an endogenous origin and the pathological picture is not typical of a tubercular lesion. The many theories advanced as to the ætiologic factor is in favor of tuberculous toxæmia. Intestinal intoxication appears to be the next most likely cause. Local treatment is important but it should be secondary to constitutional treatment. The disease should be considered as tuberculous and treated as such until the ætiology is definitely proven. Tuberculous treatment does not necessarily mean the employment of tuberculin therapeutically in all cases, but its judicious use after proper dietary, hygienic and local treatment has proven inefficacious.

ALLING.

FUNAISHI (150, **Treatment of phlyctenular inflammation of the eye**) produces surprisingly beneficial effects in seven cases by injecting intramuscularly fresh and aseptic cow's milk which has been boiled for a few minutes. The dosage is from 1 to 4*cc*, depending on the age and general condition of the patient. The injections are repeated every three or four days.

ALLING.

BLANCHARD (145, **Chancre of the conjunctiva of the upper lid**) reports the case of a woman 30 years old who complained of a feeling of heaviness in the left upper lid. Externally there was a swelling resembling a chalazion in the outer half of the lids. The preauricular, submaxillary and cervical lymphatic glands were enlarged and hard. On ectroversion of the lid one could see between the margin of the lid and the retrotarsal fold a slightly elevated, oval, very red erosion, about ½ *cm* in circumference, the margin of which seemed turned in. Its base felt very hard. The eye was unirritated and presented no pathological condition. The spirochæta pallida was demonstrated in the secretion. There was no mixed infection with pneumococci and streptococci, such as is found in ulcerated chancres. The differential diagnosis excluded glanders, Parinaud's conjunctivitis diphtheritic tarsitis, tuberculosis of the tarsus, epithelioma, chancroid, mucous patch, vaccine pustule, syphilitic tarsitis, gumma,

sporotrichosis, chalazion, herpes, and the impetiginous and pruriginous blepharitides. The manner in which the infection was transmitted is uncertain.

DENTI (147, **Chancre in the upper conjunctival fornix**) met with this lesion in a woman 44 years old. The infection came from a 4 months' old child suffering from congenital syphilis, of which she was taking care. In the median portion of the upper retrotarsal fold was a reddish induration as large as a lentil, covered with a pseudomembrane, considerable irritation of the surrounding tissues, and a hard, painless swelling of the preauricular gland. Under appropriate treatment it disappeared, leaving no trace.

ZIEGLER (172a, **The surgery of trachoma**) describes the practical problems in trachoma. The complications are persistently maintained by two mechanical processes, (1) lid friction, from blepharophimosis and trichiasis and (2) lacrimal maceration, from perversion of the tears and obstruction of the duct by trachomatous invasion. These conditions may be remedied by either conservative or radical surgical treatment. The conservative surgery of trachoma calls for (a) canthotomy and (b) rapid dilation of the duct combined with (c) galvanocautery puncture, (d) Knapp's roller operation and (e) freezing by carbon dioxide snow; while (f) galvanocautery peritomy may be used to relieve pannus. The radical surgery of trachoma requires, in addition, (g) Burow's splitting of the cartilage or (h) Kuhut-Heisrath's tarsal excision. YUDKIN.

ALVIS and WEINER (144a, **Treatment of trachoma by intramuscular injections of mercury**) report favorable relief from trachomatous lesions in a comparatively short period where mercuric salicylate is given intramuscularly in five cases which showed no improvement by the usual therapeutic agents for this lesion. YUDKIN.

HOWARD (154, **Rôle of the epithelial cell in conjunctival and corneal infections**) using the technic devised by LINDNER concludes as did the latter that the gonococus, pneumococus, Koch-Weeks bacillus and influenza bacillus exercise the pathogenic rôle of a parasite whereas the diplobacillus of Moax-Axenfeld is pathogenic saprophyte. The staphylococcus, and xerosis bacillus act as non-pathogenic saprophytes. The parasitic bacteria of the conjunctiva grow in a dense-like crust

formation on the surface of the epithelial cells. They not only phagocyte bacteria but they digest them in the same manner as do leucocytes. The ectotoxins of the parasitic bacteria diffuse into the tissue beneath them and produce a dissolution of the intracellular and interlamellar, cement substance of the conjunctival epithelium thereby permitting the bacteria to grow in the lower strata of cells. The older cells die off and the younger epithelial cells carry on the phagocytosis.

He also demonstrates that trachoma, inclusion blennorrhea and vernal catarrh have inclusion bodies which are parasites of the cytoplasm of the living epithelial cells which ingest them. The bodies are not killed by the cells, but proliferate within them until all the cytoplasm is consumed by their activity. In order to show the exact relationship of the bacteria and the inclusion bodies to the epithelial cells as they exist in the living tissue, the wet method of fixation must be employed. ALLING.

GIFFORD (151, **Marginal dystrophy of the cornea**) believes that there can be no doubt that the condition is degenerative in origin, the inflammatory symptoms observed in about half the cases being secondary and possibly due to cellular necrosis in the cornea. The small, discrete, grayish or yellowish opacities, possibly represent such areas of necrosis or deposits of degenerative products in the cornea. The possibilities of treatment are confined to careful correction of the refractive error and a mild antiseptic collyrium for constant use to protect against secondary infection. ALLING.

The pathological aspect and the clinical characteristics of epithelial dystrophy have been reported but no definite ætiologic factor has been ascribed to the manifestation.

GIFFORD (151a, **Epithelial dystrophy of the cornea and its relation to endothelial dystrophy**) describes two cases which correspond closely to Fuch's classical picture and also show the endothelial elevations described by Graves and the Friedenwalds. From his case reports it seems likely that both surfaces are affected alike and hence the same etiology, namely a vasomotor disturbance causing edema or altered local metabolism. Either surface may be effected alone.

YUDKIN.

WHITHAM (170, **Epithelial dystrophy of cornea and ocular manifestations in diabetes**) saw two cases, however, which improved with proper diabetic diet and insulin. He suggests that this corneal lesion may be of diabetic origin. ALLING.

DE GONVEA (153, **A rare case of bilateral pannus of idiopathic origin**) observed in each eye of a woman 44 years old a superficial pannus and diffuse corneal opacities with central maculæ *2mm* in diameter. In the palpebral fissures were thickenings of the epithelium looking like opalescent nodules arranged horizontally. Similar nodules were to be found in the upper halves of the corneæ. Bacteriological examinations of scrapings revealed diplococci and diplobacilli of the type of the Morax-Axenfeld which grew on ascites-agar. The writer thinks that there was at first a diplobacillus conjunctivitis and that the microörganisms were driven through the corneal epithelium by rubbing.

KEY (155, **Congenital zonular opacity of the cornea**) describes a case in which yellowish brown granules appeared under oblique illumination before the Zeiss binocular loupe, but as almost snow-white cell-like bodies under focal illumination before the slit-lamp microscope. These were in the anterior layers of the cornea. This area of opacity is surrounded by an opaque line. The condition is monocular and he believes it to be embryonic in origin and analogous to congenital zonular opacity of the lens. ALLING.

BEDELL (144b, **Band keratitis**) describes a primary and secondary involvement of the cornea. The primary type is merely a superficial process which does not involve Bowman's membrane. The film can be removed very readily with ofttimes the restoration of vision. The secondary one is usually an aftermath of ocular inflammation due to serious disease or degenerative changes in other parts of the eye. This lesion should not be subjected to operation. These corneal disturbances can be readily differentiated by slit-lamp examination.

YUDKIN.

KOYANAGI (156, **A new treatment for staphyloma**) recommends the injection into the vitreous of a ten per cent. salt solution. After about twenty-four hours the tension of the eye decreases, after a few days a decrease of the staphyloma,

and finally the eyeball becomes sufficiently atrophic to permit the use of a prothesis. In many cases the injection has to be repeated a couple of times at intervals of several days, until the hypotony is permanent and of high degree.

KRAUPA-RUNK (157, **Herpes corneæ**) distinguishes four forms of herpetic disease of the cornea: (1) Keratitis vesiculosa, which covers the entire cornea of one or both eyes with very minute vesicles; (2) the herpetic corneal ulcer with gray infiltrated margins; (3) keratitis dendritica, characterized by branching, superficial little ulcers of the cornea, and (4) the herpetic form of keratitis disciformis. In all of these types the sensibility of the cornea is reduced or absent, all forms tend to become infected secondarily by conjunctival parasites, to secondary disease of the uveal tract, and to glaucoma.

The paper by PATON (163a, **The trigeminal and its ocular lesions**) deals with the three types of ocular lesion with which the trigeminal nerve is associated, namely, herpes simplex corneæ, herpes zoster ophthalmicus and neuropathic keratitis. In the first portion of the paper Paton reviews in great detail the anatomy and physiological constitution of the nerve. Coming to the pathological side he points out that in a sense the fifth nerve is involved in all ocular pain and in consequence he divides the ocular conditions into three classes in all of which the lesion is directly affecting the nerve in some part of its course. The divisions are as follows: (1) Where the peripheral nerve terminals alone are affected. (2) Where the peripheral nerve terminals and the nerve in general are affected. (3) Where the primary lesion is central or in the ganglion. But even this classification is unsatisfactory as many of the conditions *e.g.* dendritic ulcer of the cornea, might be included in more than one of the divisions. Under the heading (1) Paton includes Recurrent Erosion of the Cornea, Superficial Punctate Keratitis, Marginal Dystrophy of the Cornea and Herpes Corneæ. Herpes Corneæ, manifested most typically as dendritic ulcer of the cornea, is either a local reaction of the cornea or of the cornea in part with a general systemic reaction to the herpes virus, also often concomitant with a general lowering of tissue resistance caused by some other disease as influenza, pneumonia, etc. The virus of the herpes cornea is next discussed in detail and

the possible relationship to the virus of varicella and encephalitis lethargica alluded to. Under heading (2) Paton discusses Herpes zoster ophthalmicus. He makes a clear distinction between herpes simplex corneæ and herpes zoster ophthalmicus. He also separates herpes zoster into 2 categories. (1) epidemic zoster (2) symptomatic zoster. Epidemic zoster is a definite infection running a routine course in most cases. Symptomatic zoster is due to a secondary involvement of the sensory nerve or posterior root ganglion in the course of some other lesion *e.g.* syphilitic meningitis, etc. A point of difference for diagnosis between the two forms of zoster is that in symptomatic zoster the neuralgic pains may precede the herpetic eruption by a long period whereas in epidemic zoster this period is not usually longer than 1 to 4 days. It is rare for other than the ophthalmic division of the trigeminal to be affected and often the lacrimal and naso-ciliary branches of this division of the nerve escape. Attention is called to the original statement of Hutchinson that only when the naso-ciliary nerve is involved is the globe itself affected but it is also pointed out that exceptions to this general rule occur. The symptoms and signs of zoster are discussed in detail. With regard to the occurrence of optic neuritis in zoster Paton has seen two cases. The differentiation given between herpes zoster and herpes febrilis is interesting.

Herpes Zoster.	Herpes Febrilis or Simplex.
1. Follows distribution of nerve or nerves affected.	May do so; but may be irregular in distribution.
2. Always a preceding neuralgia and arises independently of and preceding disease.	No preceding neuralgia and follows on many infections of different kinds producing lowering of tissue resistance.
3. Attacks true skin and leaves permanent scarring.	Only superficial dermis affected. No permanent scarring.
4. One attack seems to confer immunity.	Tending to recurrences.
5. 3–4 weeks duration with long period following of diminished sensibility.	Duration short, no improvement of sensibility.

Also, it has been found that the sera or antigens of zoster patients do not give positive complement fixation tests against

antigens or sera from cases of herpes febrilis. The typical corneal lesion of herpes febrilis is the dendritic ulcer, whereas in zoster the usual primary lesion is multiple small round infiltrations in the superficial layers of the substantia propria of the cornea which may give rise to vesicles, but often do not, or in keratitis profunda. The close relation between zoster and varicella is discussed.

Neuropathic keratitis: Paton's experience of this condition is for the most part from cases of trigeminal neuralgia treated either by Gasserectomy or alcoholic injections into the ganglion within a few days of the operation or injection. The whole corneal surface may become stippled and hazy and later secondary infection may occur. If the lids are sutured together before secondary infection has become well established, the cornea will clear up rapidly. Paton calls attention to the extraordinarily good response of corneæ, affected in this way, to lid suture and to the fact that any tendency to relapse can be prevented by even the smallest band of adhesion between the lids. The pathogenesis of this condition is discussed at length. The various hypotheses (the trophic, vasomotor, traumatic evaporation and bacterial infection), are reviewed at length, and Paton comes to the conclusion that no one of them is sufficient to explain the phenomena in its entirety. DOYNE.

KREIKER (158, **Observations with the slit-lamp on the development of vascularization in the cornea**) claims that the formation of vessels in the cornea is through the pushing forward of the vessels of the limbus, the loops of which become longer and broader. Anastomoses take place through the touching of loops one upon another, or of two branches of one and the same loop. The first sign of retrogression is a diminution of the quantity of blood in the vessels; one may perceive stasis or emptiness in the capillaries. The mechanics of a retrogression covering several weeks may easily be observed within half an hour if adrenalin is instilled.

From a study of some 370 cases, GUY (153a, **Interstitial keratitis in congenital syphilis**) concludes that treatment should be continued long after the eye complication is cleared, and should be intermittent to avoid the development of arsenic and mercury fast strain of spirochetes. Iodides should

be used for their resolvent effect because of the tendency to fibrosis in congenital syphilis and the gummatous nature of interstitial keratitis. The general physical condition of the patient should be improved by medical and physical treatment. YUDKIN.

From an experimental study on rabbits, KEY (155b, **Influence of protein therapy on experimental staphylococcus infection of the rabbit's cornea**) finds that there is no important difference between the effect of anti-diphtheritic serum, concentrated horse serums and typhoid vaccine upon the local infection which he introduced by injecting staphylococcus pyogenes aureus in the layers of the cornea. He notes that a strain of micro-organisms taken from the eye are more virulent than that from infections at different parts of the body. Clinically he uses anti-diphtheritic serum in small doses of 3 to 4 c.c. (2400 to 3200 units). This dosage is harmless yet sufficient to produce moderate systemic reaction just short of anaphylaxis. The positive systemic reaction to protein injection is a valuable therapeutic measure.

YUDKIN.

From an analysis of the literature, KEY (155a, **Brittle bones and blue sclera**) finds that the blue sclera is the only feature of true (fragilates ossium) hereditary hypoplasia of the mesenchyme which is present in every case. This hereditary factor is a dominant one and there is no skipping of generations. The blue appearance of the sclera is not due to a deposit of pigment but is probably due to an abnormal transparency of the fibrous tissue which permits a shining through of the deeper layers. The fractures in these cases are not spontaneous but are due to definite causes. The deformities are due to malnutrition of old fractures and not to softening of the bone. The hypoplasia seems to be an anomaly of development rather than a derangement of metabolism and is apparently limited to those tissues derived from the mesenchyme. YUDKIN.

PILLAT (165, **Metastatic keratitis after epidemic cerebrospinal meningitis**) found a deep seated disease of the cornea of each eye in a 6-year-old child suffering from epidemic cerebrospinal meningitis. The right eye was blind, the vision of the left reduced to counting fingers at half a meter. As he

could perceive no other cause for the deep-seated corneal infiltrates, he believed them to be metastatic.

SCHWARTZ (167, **Nutritional keratomalacia**) reports a case of a child 10 months old, in which the outstanding symptoms were stupor, irritability on being disturbed, weight loss, pallor, doughy skin, dry inflamed mouth, cracked lips, fever and characteristic corneal lesion. The patient recovered following the administration of cod-liver oil, given by mouth and hyperdermically, and a diet of whole lactic acid milk.

ALLING.

VERHOEFF (169, **The cause of keratitis after Gasserian ganglion operations**) considers that lacrimal secretion cessation after Gasserian ganglion operation is probably due to injury to the great petrosal nerve, a branch of the seventh, and that the keratitis sometimes arising from this deficiency of lacrimal secretion is an exposure keratitis. ALLING.

WOLZ (171, **Hereditary nature of keratoconus**) presents six new cases of familial or hereditary keratoconus. A number of such cases have been recorded. It has been met with in brothers and sisters. In one case it possibly existed in the grandmother on the mother's side of two sisters. It has been found in mother and daughter in father and son, in two uncles, in a niece and an aunt. Several times the keratoconus was more marked on one side than on the other in members of the same family. The heredity seems to be not only of the collateral, but also of the direct and indirect form.

GARRETSON AND COSGROVE reported (150a, **Ulceration of the cornea due to bacillus pyocyaneus**) an epidemic of sixteen industrial cases in the same factory. They found the strain of bacteria a very virulent one, and with the aid of the staphylococcus aureus invaded the already injured cornea producing ulceration and destruction of the eye. Experimental investigation of the affect of these bacteria on rabbits' corneæ revealed the fact that the tissue was not very susceptible to the invasion of the bacillus pyocyaneus alone. The injection of saline suspensions of dead bacteria and broth filtrates introduced an immunity against the bacillus pyocyaneus.

YUDKIN.

YOUNG (172, **Electrothermophore in the treatment of corneal ulcers**) reports favorable results in twenty-five cases

of various types of corneal ulceration treated with Shahan thermophore. He finds the thermophore is practically a specific in the treatment of infected ulcers, relieving pain, stopping progress of the ulcer and insuring the best possible visual results. ALLING.

X.—THE LENS.

173. ADDARIO. **The artificial ripening of cataract.** *Bolletino d'oculistica*, October, 1922.

174. ASMUS, E. **Infection after discission of after cataract.** *Zeitschrift f. Augenheilkunde*, li., January-February, 1923.

175. BULSON, ALBERT E., JR. **Prolapse of iris after cataract extraction.** *Journal of the A. M. A.*, lxxxv., p. 322.

176. CHARLIN. **A sign of cheer to patients recently operated on for cataract.** *Annales d'oculistique*, clx., 8, p. 629.

177. ELSCHNIG, A. **Detachment of the zonular lamella in glass blowers.** *Klin. Monatsbl. f. Augenheilkunde*, lxix., 1922, and lxx., 1923.

177a. FOX, L. WEBSTER. **Congenital ectopia lentis.** *Amer. Jour. of Ophth.*, ix., 257, 1926.

178. FRANKLIN, WALTER SCOTT, and CORDES, FREDERICK C. **Electric cataract.** *Journal of the A. M. A.*, lxxxv., p. 244.

179. GIFFORD, SANFORD R. **Allergic and toxic properties of lens protein.** *Ibid.*, lxxxv., p. 351.

180. v. HORVATH, BELA. **Formation of cataract after influenza.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., 1923.

181. JACKSON, EDWARD. **Progress of senile cataract under hygienic care.** *American Journal of Ophthalmology*, October, 1924, p. 775.

182. KUBIK, J. **Detachment of the zonular lamella in glass blowers.** *Klin. Monatsbl. f. Augenheilkunde*, lxx., 1923.

183. LYLE, DONALD J. **Babbitt metal in the lens without lenticular opacity.** *American Journal of Ophthalmology*, November, 1925, p. 887.

183a. MOORE, FOSTER R. **The Corneal Section in Cataract Extraction. A Small Maneuver.** *British Journal of Ophthalmology*, Jan., 1927.

184. STARK, H. H. **Asteroid hyalitis.** *A. J. O.*, October, 1924, p. 770.

185. VALOIS and LEMOINE. **Remarks relative to lavage of the lenticular sac in the course of an operation for cataract. Artificial reformation of the anterior chamber.** *Annales d'oculistique*, clx., 1, p. 28.

186. VOGT, A. **New observations concerning human lenses with double focal point.** *Zeitschrift f. Augenheilkunde*, March and April, 1923.

186a. WÜRDEMANN, HARRY V. **Expulsive choroidal hemorrhage after cataract extraction.** *Amer. Jour. of Ophth.*, ix., 427, 1926.

187. YOUNG. **On macular perception in advanced cataract.** *British Journal of Ophthalmology*, April, 1923.

By slit-lamp examination, FOX (177a, **Congenital ectopia lentis**) observed fibers running straight and apparently taut

from the normal zonule to and into the margin of the dislocated lens. From his findings in two cases he agrees with Treacher Collins that a certain proportion of cases are due to arrested growth of the lens and its failure to completely fill the space allotted to it. YUDKIN.

ADDARIO (173, **The artificial ripening of cataract**) recommends discission of the anterior capsule in cases of unripe or nuclear cataract. In every case a preliminary iridectomy should be performed at least two weeks before. He claims that at the extraction the entire cloudy lens usually follows the knife, and that if any of it remains behind it can be removed by means of a broad spatula. In this way he says that he has avoided the formation of secondary cataract for ten years.

FOSTER MOORE (183a, **The corneal section in cataract extraction**) describes this maneuver which he has devised to prevent buttonholing the iris when making the corneal section in the extraction of cataract. It occasionally happens to everyone that, when cutting out, the iris falls over the edge of the knife and an involuntary iridectomy is done. This is more especially liable to happen when the lens capsule is incised between puncture and counter puncture.

To describe the proceedure in the author's words—"If, in such a case (Iris having fallen over the edge of the knife), a repositor is taken in the other hand whilst the knife is in position, it will be found that, by a stroking movement of the repositor over that part of the cornea which overlies the blade, the iris can be expressed from between the blade and the cornea, and made to slip under the knife, and so the section completed without damage to the iris."

Foster Moore says that he has done this on several occasions and very seldom found it to fail.

DOYNE.

ASMUS (174, **Infection after discission of after cataract**) performed 20 out of 315 discissions through the cornea, 295 through the sclera. Of the first group of 20, four suffered from infection and one eye was lost. Of the second group of 295, four likewise suffered from infection and one eye was lost. This showed that in his hands the subconjunctival-scleral introduction of the knife is the safer. But, he says,

there are cases in which the most experienced operator must enter through the cornea and take the chance of infection. Yet he seems to have taken utmost precautions. The operations were performed in the hospital, not in the clinic, in all the infected cases. The knives used were Kuhnt's, Berheimer's, and Knapp's. Apparently clean eyes were washed out with a ½% solution of zinc sulphate; if no flecks of mucus appeared the operation was performed, otherwise a bacteriological examination was made. Chemical or chemotactic measures are not suited to prevent the access of infection.

BULSON (175, **Prolapse of iris after cataract extraction**) finds that post-operative prolapse of the iris is less likely to occur in those cases in which the combined operation has been performed, particularly when a large conjunctival flap has been made and in which the wound has not been submitted to unnecessary and meddlesome early inspection. In the event of a prolapse of the iris which is not well covered by a conjunctival flap, a delay of from five to six days after its development offers the advantage of a firmer healing of the wound outside of the area involved and an excision of the iris may be made more safely and effectively. He does not recommend reduction with the actual cautery. Whether the prolapse is excised or reduced by cautery, the area should be covered by a Kuhnt conjunctival flap. ALLING.

From a critical study and his own operative experience, WÜRDEMANN (186a, **Expulsive choroidal hemorrhage after cataract extraction**) finds that bleeding may occur in any operation that involves opening of the eyeball. The exciting cause is anything that brings strain on the operated eye such as sudden decrease of ocular pressure, venous hyperemia from vomiting, and strain of coughing. The blood invariably comes from a ruptured choroidal vein. Microscopic examination of such eyes shows hyaline degeneration of the retina, the vascular walls of the uvea partly thickened, liquid vitreous and the retinal vessels very narrow. YUDKIN.

CHARLIN (176, **A sign of cheer to patients recently operated on for cataract**) revives the old custom of having a patient, at the first change of dressings at the end of twenty-four or forty-eight hours, look at the face of the surgeon, which appears blurred, and then have him suddenly see the face

clearly by the interposition of a +13D. lens. This cheers the patient, for it is an unexpected and positive demonstration to him that he may expect a good result.

ELSCHNIG (177, **Detachment of the zonular lamella in glassblowers**) believes that he has seen by means of the slit-lamp this lesion in three eyes of two elderly glassblowers. In the first case he saw on the anterior surface of the lens a polygonal, extremely thin membrane as clear as water, glistening yellow at the margins, which trembled slightly with the movements of the eye and were slightly rolled in, especially above, so that the entire structure apparently was attached to the lens only in its middle and was concave toward the anterior chamber. The lens presented an immature cataract in the pupillary region. When the lens was later extracted an attempt was made to seize this membrane with forceps, but it failed. After recovery it was no longer visible. In the second case a glassy membrane with some pigment granules lay on the anterior capsule. It was rolled in at the sides and slightly folded backward below, so that it was apparently attached to the capsule only above. The structure was slightly shoved together below when the pupil was contracted. Above the slightly fluttering membrane became invisible and lost in the anterior capsule. The shagreen of the epithelium of the anterior lens capsule was distinct. The lens was almost perfectly clear. In certain lights the margins had a golden reflex. The first patient was 52 years old and said he had worked in glass of a high melting point eight hours a day for forty years. The second was 50 years old and had worked in the same way for 38 years, formerly twelve, now nine hours a day. Elschnig is inclined to refer these lesions to the prolonged exposure of the eyes to the effects of heat.

KUBIK (182, **Detachment of the zonular lamella in glassblowers**) confirmed the findings of Elschnig by the demonstration of similar conditions in three glassblowers aged 46, 55, and 61 years. In three other patients, the youngest of whom was 37, and had worked 23 years as a glassblower, he found fine lines, which he took to be ruptures of the zonular lamella and probably the starting points of detachments.

v. HORVATH (180, **Formation of cataract after influenza**) reports a case in which he thinks there was a connection be-

tween the attack of influenza and the development of the cataract. The patient was a young woman 24 years old who had an attack of influenza with much headache which lasted three weeks. On the second day of the disease exophthalmos and a swelling in the parotid region appeared, later a purulent conjunctivitis. Then these troubles disappeared. In the second week of the disease the vision of the right eye became bad. Two months later a cortical cataract was found to be present.

FRANKLIN and CORDES (178, **Electric cataract**) report a case and a survey of literature. They find that in dealing with electrical burns about the face and eyes, serious changes in the globe may take place without producing lenticular opacities. The eye nearest the site of the burn usually shows the most marked changes. When cataracts are found, they are rather characteristic. The cortex and the deeper layers may be involved, and the opacities as a rule are flaky, but at times may be finely granular. ALLING.

LYLE (183, **Babbit metal in the lens without lenticular opacity**) reports a case of foreign body in the lens which can be readily seen under mydriasis two years after injury. There is very little, if any, opacity surrounding the foreign body and the vision is not impaired. ALLING.

JACKSON (181, **Progress of senile cataract under hygienic care**) finds that the progress of cataracts is usually slowed and often permanently arrested when the following conditions have been observed: (1) Avoidance of eye strain by proper change of glasses whenever required; good conditions of illumination; and limited periods and maintenance of proper distance for near work. (2) Preservation of general health and nutrition, including the taking of sufficient water in the daily diet, and (3) the proper treatment of ocular diseases that may be associated with lens opacities, particularly uveal inflammation. ALLING.

YOUNG (187, **Macular perception in advanced cataract**) takes the patient into a dark room and places a large dark disk with a round hole in its center directly before the eye, the hole being exactly opposite the center of the pupil. An electric lamp is placed behind the disk and the patient is asked if he can perceive the light. If he does, the macula

is surely intact. Yet it hardly excludes with certainty a disease of the macula.

VALOIS and LEMOINE (185, **Remarks relative to lavage of the lenticular sac in the course of an operation for cataract. Artificial reformation of the anterior chamber**) emphasize the advisability of restoring the anterior chamber artificially with a fluid as nearly as possible of the same composition as the aqueous at the close of the operation. The advantages, they claim, consist in a quick healing and cicatrization with almost no inflammatory reaction and very slight postoperative pain. The consequences of a lowered tension of the eye, due to the escape of the aqueous, such as hyperæmia of the iris and ciliary processes, dilatation of the retinal vessels, and œdematous infiltration of the cellular tissues, are reduced to a minimum. Another factor worth considering is the composition of newly formed aqueous, which contains a considerably more than normal quantity of albumin and coagulates spontaneously, so that it may almost be considered as pathological, being formed in great part of blood serum and fluid resembling that of exudations. Finally the almost immediate replacement of the contents of the anterior segment of the eye, very nearly completely emptied during the operation, by the artificial restoration of the anterior chamber prevents, or at least diminishes markedly, the manifold disturbances met with in the course of healing. In addition the individual parts in the anterior segment of the globe may best be brought to lie in a good position by this means.

GIFFORD (179, **Allergic and toxic properties of lens protein**) found 12.6 positive dermal reactions to lens protein in eighty-six patients tested. Six of these positive cases gave an unusual post-operative ocular reaction which they interpret as due to their sensitization to lens protein. Eight patients who gave negative skin tests showed an ocular disturbance which they thought was probably due to absorption of the lens matter. They note that normal lens matter shows toxicity when injected into the anterior chamber of experimental animals and that cataractous lens matter was apparently more toxic. They believe that the chemical differences between them explain the toxicity. There is evidence in the ophthalmic literature that autolysis of lens protein occurs

with the production of leukomains and allied products, which may be toxic. ALLING.

VOGT (186, **New observations concerning human lenses with double focal point**) had the opportunity to examine five lenses with double focal point with the slit-lamp. The lenses were in three persons, a man 62 years old, a man 65 years old, and a woman 80 years old. He found in these lenses a diffuse cataract of the embryonal nucleus which was invisible by the usual methods of examination, or threw only an unimportant shadow. It was only the central, embryonal portion, not the entire nucleus, which was involved in the change of index. The double focus is not caused by a diminution in the index of the cortex, as has been surmised, but by an increase in the index of the nucleus. Should the entire nucleus be involved the difference in the refraction between the cortex and the center would not be demonstrable, even when the pupil was widely dilated.

STARK (184, **Asteroid hyalitis**) reports a case in which the bodies developed while under observation. The patient had a tuberculous lesion and was treated with tuberculin. The writer concludes from his observation, that the asteroid bodies are probably an exudate secondary to a tuberculous cyclitis. He believes that the earlier they are discovered and treated, the greater the possibility of their absorption, but should they remain unabsorbed for a sufficient length of time, they become calcified. ALLING.

(*To be continued.*)

NOTICES.

Ernst Fuchs Fund.

Ophthalmologists for many years have received training and instruction in Vienna. The influence of the Vienna school of ophthalmology is world wide and its clinical teaching has been of inestimable value, especially in developing ophthalmology in this country.

Unfortunately, owing to the present economic conditions in Austria, the position of the younger ophthalmologists in Vienna,—for the most part, assistants in the two University clinics of ophthalmology in Vienna,—is a very difficult one. It seemed, therefore, that an opportunity to help the Vienna ophthalmological school and to preserve its tradition would appeal to those who have had the advantage of studying there.

For this purpose, it is proposed that a fund be collected which will carry the name of ERNST FUCHS and which will have the following purpose:—

To give financial aid in experimental investigations.
To help defray the cost of publications, and
To furnish money for attending congresses.

This fund is to be managed by the directors of the two University Eye Clinics in Vienna and by the chairman of the Vienna Ophthalmological Society.

Will you demonstrate your sympathy in this appeal by becoming a subscriber to this Fund?

Communications should be addressed to Dr. E. V. L. Brown, Secy., 122 South Michigan Boulevard, Chicago, Ill.

Adolph Barkan, Chairman.
Arnold Knapp,
George S. Derby,
E. V. L. Brown,
Joseph M. Keller.

CORRESPONDENCE.

BERLIN N. W., May 18, 1927.
HAENDELSTR. 21.

THE EDITOR,
Archives of Ophthalmology.

DEAR SIR:

In the translation in *Clinique ophtalmologique*, April, 1927, of my article which appeared in the *Archives of Ophthalmology*, November, 1926, an error has unfortunately occurred. It is stated that in "almost all cases" the operation for acute glaucoma can now be avoided. This, of course, is wrong. What I wished to say was that "in a group of cases" the operation can be avoided; I have had failures with the medicinal treatment.

On the other hand, a miotic which is capable of reducing an atropinized pupil rapidly and for a number of hours must be the subject of further experimentation, even if no other result is attained than the avoidance of operations in a certain percentage.

Very truly yours,
DR. CARL HAMBURGER.

VIII.—**Applied Refraction,** by DR. HOMER E. SMITH, New York. 131 pp., with 9 figures in the text. 1927. William Wood & Co., New York. Price, $2.75.

This book does not deal with the elements of physiological optics nor does it give a description of ordinary refractive methods, but presupposes a knowledge of these subjects. It is therefore not a treatise on refraction in the ordinary sense, but rather takes up under 24 brief chapters, the chief difficulties that the trained refractionist is apt to meet with in his daily work, explains the reasons which underlie these difficulties, and points the way out. Probably a better title for the book would be, "Refinements in the Art of Refraction." It is plain that it was not designed for the beginner in refraction, but for the physician of considerable experience in this line of work, but who still occasionally meets with conditions that are puzzling. To such it should be a valuable asset. The book is well written in the terse, lucid style that is characteristic of the author. If a book can ever be said to bear a likeness to its author, this one certainly resembles its genial parent. Even the frequent attempts at subtle humor will not seem out of place to those who have known him. In place of the usual bibliography a unique but appropriate epilogue is appended.

A. H. T.